THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 86 DECEMBER, 1961 No. 6

THE PERFORMANCE OF SCREEN INTENSIFICATION AND CINEFLUOROGRAPHIC SYSTEMS*

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IT IS a great honor and privilege to speak to you today on the occasion of the Annual Oration of the Philadelphia Roentgen Ray Society. It is appropriate that this year's oration has been dedicated to the distinguished engineer, Mr. H. Clyde Snook. Technologic advancement in the field of Radiology has never been more rapid than in the last few years. Yet, such progress has been possible only with the sound physical and engineering foundation which men like Clyde Snook laid for Radiology during the early period following the discovery of the x-ray. His tireless efforts to develop and build apparatus which had practical usefulness to the radiologist opened the way for physicians everywhere to reap the enormous benefits of the x-ray.

I am indebted to Mr. Charles Smith and to Dr. Richard Chamberlain for much of the information I have to tell you about Mr. Snook. Dr. Herbert Stauffer has kindly given me valuable data as well.

HOMER CLYDE SNOOK: A HISTORICAL SKETCH

Homer Clyde Snook was born in 1878 in Antwerp, Ohio, the son of Wilson H. and Nancy Jane Snook. After the completion of his primary education in the public schools of that city, Snook became a student at Ohio Wesleyan University and received his A.B. degree there in 1900. At the time Röntgen made his discovery of the x-ray, Snook was a student of Professor W. G. Hormell in the university's Department of Physics. Because both were enormously interested in Röntgen's discovery, they at once began experimental work to produce x-rays in their laboratory. Following a period of several days of intense activity, activity which rarely has been paralleled, Snook and Hormell were successful in putting together a system which indeed generated x-rays.

Although Snook taught for a short time at Allegheny College after his graduation, much of his life was spent in Philadelphia. Snook's decision to move to this city was fortuitous for there is little doubt that much of his success came as a result of the environment provided by this outstanding center of Clinical Radiology. Snook not only had the opportunity to test his ideas with many of the world's finest radiologists, but of even greater importance he

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Presented as the H. Clyde Snook Annual Oration at The Philadelphia Roentgen Ray Society, November 10, 1960.

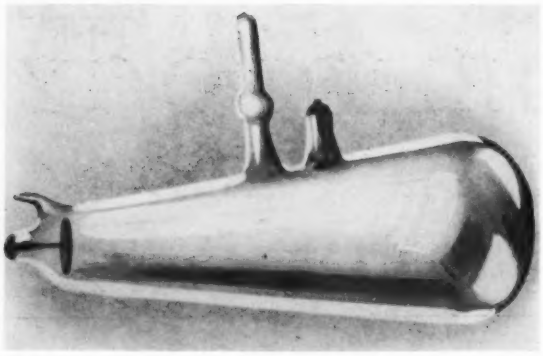

Fig. 1. The Hittorf tube.

received from these men a scientific stimulation seldom equaled anywhere.

The story of Snook is inseparable from the story of Radiology during the first two decades of this century. The source of the x-radiation which Röntgen discovered and which Snook soon duplicated in Ohio was a tube variously known at different times as the Geissler tube, the Plucker tube, the Hittorf tube, and the Crookes tube. All were essentially the same in basic design and were constructed for the study of the characteristics of electrical discharges in gases. These tubes had a cathode and an anode (Fig. 1). The anode, however, was in such a position that when the gas was ionized within the tube by the presence of a high potential across the two electrodes, electrons were projected from the cathode on the glass at the far end of the tube. The electron bombardment of the glass caused it to fluoresce with a green color. Although these tubes had been used as far back as 1859 by Plucker, it remained for Röntgen to discover that, in addition to green fluorescence, x-rays were also produced by the electron-glass interaction. Interestingly, at the time of Röntgen's discovery, the attention of more than one American physicist was being directed toward the inexplicable fogging of unopened packets of photographic plates placed in the neighborhood of a Crookes tube.

Professor Röntgen did not discover the x-ray by accident. As related in an anonymous account written for the Snook-Roentgen Manufacturing Company of Philadelphia in 1916, Sir James Mackenzie Davidson told Mr. Snook his recollections of an interview with Professor Röntgen in Würzburg shortly after the discovery of the rays. Sir James stated that he asked the question, "What were you doing with the Hittorf tube when you made your discovery?" "I was looking for invisible rays," replied Röntgen. "What made you use a barium platino-cyanide screen?" "In Germany we use it to reveal the invisible rays of the spectrum, and I thought it a suitable substance to detect any invisible rays the tube might give off." The sudden shining of that screen led to one of the greatest scientific discoveries of history.

In the twenty to thirty years following the discovery of the x-ray, Snook played a vital role in technologic developments in the United States. Although he is known for many things, Snook's greatest contribution undoubtedly was the so-called interrupterless x-ray transformer shown in Figure 2. For the first dozen years after the discovery of the x-ray, roentgenologists and physicists employed induction coils (Fig. 3) and static machines (Fig. 4) to develop the electrical potentials needed to produce x-rays. These instruments, however, had many disadvantages including the limited power which could be delivered to the x-ray system.

It was at this stage of the development

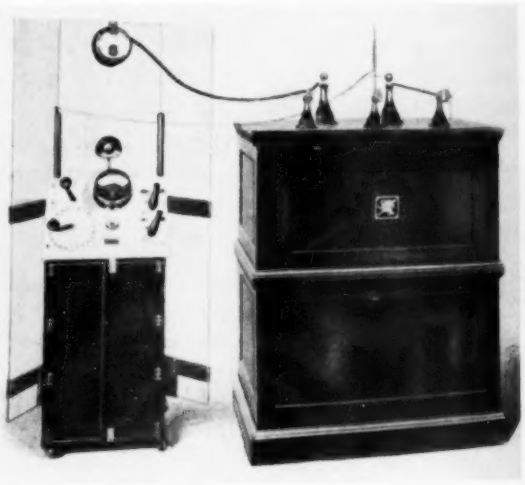

Fig. 2. The Snook interrupterless x-ray transformer.

of x-ray apparatus that Snook, together with his colleagues Herbert White and Edwin Kelley, organized the Radioelectric Company which a few months later became the Roentgen Manufacturing Company. Their first office and workshop was a second story back room at 226 Ionic Street, Philadelphia (Fig. 5). These modest quarters, which were in one of the oldest sections of Philadelphia, were destined to be the birthplace of Snook's major contribution to Radiology.

In endeavoring to find some means to prevent the inverse current from passing through an x-ray tube, Snook made a thorough analysis of the erratic performance of the induction coil by taking oscillographic readings of a coil of the Ruhmkorff type in the Randal Morgan Laboratory of Physics at the University of Pennsylvania. His report, published in January, 1907, was the first comprehensive study of the vagaries of this type of apparatus. During his investigations he devised a high-tension synchronous spark gap and from the oscillographic readings made with this apparatus conceived the idea of using rectified alternating current for exciting the x-ray tube. His first step was to replace the induction coil with a closed core transformer, after which he developed the rotating rectifying switch which became so closely identified with his name.

The experimental design and early production of the interrupterless transformer

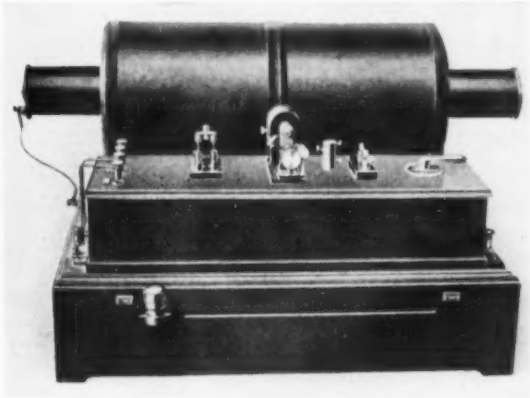

Fig. 3. The Queen induction coil.

Fig. 4. A typical static machine used in the generation of x-ray tube potentials early in this century.

was done in the little back room on Ionic Street and the first unit for hospital use was installed in the Jefferson Hospital in Philadelphia in June of 1907. After this time there followed a rapid replacement of the induction coil with the transformer. In 1908 Mr. Snook described his machine in a paper given before the International Roentgen Congress in Amsterdam. He also visited England and Germany and after that time for a number of years the interrupterless transformer was manufactured widely throughout the world.

It may be worth recording that one of the earliest transformers manufactured by Snook was sold to Baetjer at The Johns Hopkins Hospital in 1907. Historians of this conservative institution in Baltimore may someday comment on the fact that this equipment was still in operation, carrying a heavy clinical load, as late as 1946.

Snook played an important role in radiologic developments in Philadelphia. He was among the founders of the Philadelphia Roentgen Ray Society and was active in its affairs until he moved from Philadel-

FIG. 5. The birthplace of the interrupterless transformer, 226 Ionic Street, Philadelphia, Pennsylvania.

phia in 1916. Following his departure from Philadelphia, Snook became Vice-President of the Victor Electric Company of Chicago. From 1918 to 1925 he was a member of the engineering staff of the Western Electric Company. In 1925 he joined the Bell Telephone Laboratories and in 1927 he became an independent consulting engineer.

Snook was a Fellow of the American Institute of Electrical Engineers and of the American Physical Society, and a member of the American Roentgen Ray Society. He was also the recipient of many honors, including the Edward Longstreth Medal of the Franklin Institute in 1919, the Gold Medal of the Radiological Society of North America in 1923 and the Gold Medal of the American College of Radiology in 1928. He was honored by his alma mater in 1926, when he was granted a degree of Doctor of Science. H. Clyde Snook died in Summit, New Jersey, September 22, 1942.

The world owes much to Clyde Snook. He brought distinction to his profession. Although the march of progress now has left Snook and his contemporaries far behind, we should not forget the contributions made by these dedicated men, for without them we should not be where we are today.

PRINCIPLES OF SCREEN INTENSIFICATION

I should like to turn now to the field of screen intensification and cinefluorography, two areas in which Snook undoubtedly would have been intensely interested if he were still alive. In my discussion of these subjects I should like to speak first of screen intensification and to refer briefly to a number of the fundamental principles upon which it is based. Such a discussion is necessary as a prelude to my comments on cinefluorography, since without screen intensification cinefluorography would have little more than theoretic interest to the radiologist due to the excessively large radiation doses which must be used when non-intensified techniques are employed.

When the useful beam of an x-ray is projected through an anatomic or other structure, many and some times most of the photons which comprise the beam are absorbed. A few, however, pass through the structure to emerge on the other side. If these are allowed to fall on a fluorescent screen, some are absorbed and their energy is converted into light.

If all anatomic tissues absorbed x-ray photons equally, the number received by the fluoroscopic screen would be uniform from one portion of the screen to another and, hence, the light generated by the screen would exhibit no image pattern. If, on the other hand, some tissues absorbed more x-ray photons than others, the brightness of the screen would not be constant from one point to another but instead would vary in accordance with the configurations of the anatomic structures through which the radiation passes; that is, the screen would bear an image pattern. Although many tissues absorb x-rays nearly equally,

others, because of differences in density and/or atomic number do not. Therefore, the images of many anatomic components may be recorded fluoroscopically and roentgenographically.

From the description of the manner in which fluoroscopic images are produced, it will be clear that the diagnostic information recorded by a fluoroscopic screen is carried from patient to screen by the myriads of x-ray photons which comprise the x-ray beam. Indeed, each transmitted photon carries a certain small increment of diagnostic information and it is the sum of all of these bits of data which governs the total quantity of diagnostic information recorded by the screen at any instant. The amount of information provided by a screen can never be greater than that carried by the photons which impinge upon it. It, of course, can be less if such factors as fluoroscopic grain size do not permit efficient utilization of the information carried by the photons. The point that must be emphasized, however, is that the diagnostic information provided by a fluoroscopic screen can be no greater than the sum of all of the increments of information carried to the screen by the photons of the x-ray beam. What has been said for the fluoroscopic process is also valid, of course, for roentgenography and for cinefluorography.

Since each x-ray photon may be assumed to have associated with it a certain amount of diagnostic information, it follows that when an x-ray beam of high radiant intensity is projected through a patient, the detail recorded on a fluoroscopic screen will be considerably greater than when a beam of low intensity is used. In the first instance, there are a great many x-ray photons carrying diagnostic information and hence the detail of the resultant image is relatively good; in the second instance, the number of x-ray photons is smaller and hence less diagnostic information is available and degradation of image quality results. That this is true may be easily demonstrated if one fluoroscopes a patient first with an x-ray tube current of 5 ma. and then with a current of 0.5 ma., all other factors being equal in the two situations. Fluoroscopic image detail will be observed to be substantially greater when the higher x-ray tube current is used.

The foregoing discussion provides a qualitative understanding of the fluoroscopic process. For a quantitative evaluation of fluoroscopic performance, it is necessary that we turn our attention to the quantum statistics of physiologic optics.

It has been shown by physiologic experiment[4] that the performance of the eye measured in terms of its ability to record visual data is largely limited by statistical fluctuation in the number of light photons reaching the retina. Although most individuals are unaware that such fluctuation exists, it nevertheless is present to a greater or lesser degree under all viewing conditions and ultimately determines the fineness of structure that the eye can perceive. Fluctuation of course does not constitute the only factor which limits visual performance. In diagnostic radiology, such factors as the diameters of the retinal rods and cones and a large number of physical factors associated with the x-ray equipment and recording media also introduce limitations. However, these factors only contract the limits already set by statistical theory and hence are of secondary importance in a discussion where the fundamentals of data recording are under examination.

In general, the statistical fluctuation in light intensity which occurs at the retina of one's eye is proportional to the square root of the average number of photons projected on the retina within the storage time of the eye (about 0.2 second). For example, if the eye is observing an object 1 mm. in diameter at a viewing distance of 20 cm., the number of photons reaching the retina from the object during each 0.2 second will be of the order of one million at normal levels of illumination. Therefore, the average fluctuation in light intensity at the retina from moment to moment will be approximately the square root of one million; that is, the fluctuation will be of the

order of ±1,000 photons or about ±0.1 per cent of the average light intensity. Now, if the illumination of the object is reduced to levels comparable to those occurring at fluoroscopy, the effective number of photons reaching the retina within 0.2 second may be as small as 100. Accordingly, such a number will have an associated average fluctuation of ±10 photons or ±10 per cent of the average light intensity. Hence, when the illumination of an object is reduced, the per cent fluctuation in the rate at which photons arrive at the retina increases.

The effect of this fluctuation will be evident if one considers the situation which occurs when the 1 mm. object under observation is surrounded by a field whose average illumination is 5 per cent greater than that of the object; that is, the contrast between the object and the surrounding field is 5 per cent. Under such a condition, if the object is observed under normal illumination, where the fluctuation in brightness from moment to moment is ±0.1 per cent, the fluctuation will not interfere noticeably with the perception of the object. On the other hand, if the illumination of the object is at a level corresponding to that occurring in fluoroscopy where the fluctuation is of the order of ±10 per cent, the object may at one moment appear darker and at the next moment lighter than the surrounding field. Obviously, such variation will interfere markedly with the perception of the object and will create considerable confusion in the mind of the observer.

The magnitude of the statistical fluctuation which occurs from moment to moment in the number of photons received by the eye of an observer during fluoroscopy and the extent to which such fluctuations quantitatively affect image perception have been the subject of exhaustive study.[5] It has been found that the magnitude of the fluctuation is largely governed by that stage of the fluoroscopic process where the photon intensity (*i.e.*, the number of photons per unit area per second) falls to its lowest level and that the limits set by this fluctuation on the size of the smallest image which an observer can detect is given by the formula:

$$d = \frac{5}{C(nt)^{1/2}} \times 10^2 \qquad (1)$$

where d is the diameter of the smallest perceptible image in millimeters, C is the per cent contrast of the image, t is the storage time of the eye in fluoroscopy or the exposure time of the film in cinefluorography and n is the number of photons per second emerging from each square millimeter of the image during that stage of the fluoroscopic or cinefluorographic process when the photon intensity falls to its lowest level.

Figure 6 is a graphic presentation of the photon intensities which exist during the several stages of the fluoroscopic process. Stage 1 is a plot of the x-ray photon intensity prior to the beam's absorption by

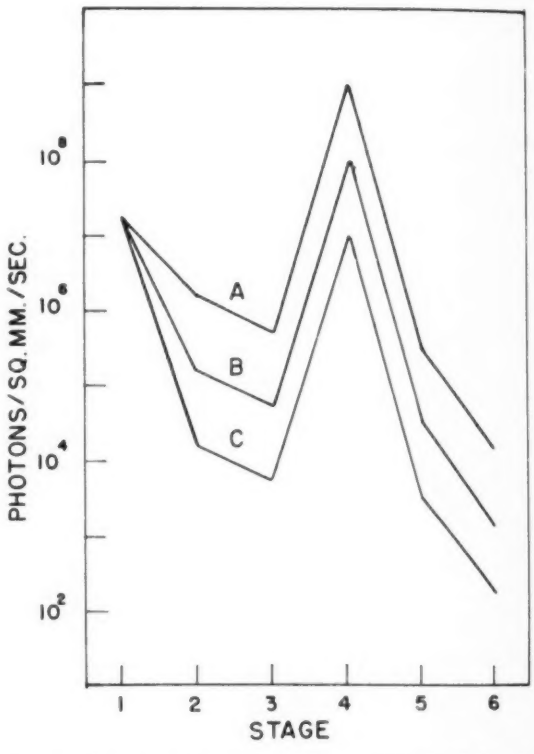

FIG. 6. Quantum diagram of the conventional fluoroscopic process during the examination of: A, the anteroposterior chest; B, the anteroposterior abdomen; and C, the lateral lumbar spine.

the patient under examination. The data are for a fluoroscope operating at 90 kvp., and 3 ma., with 3 mm. of aluminum filtration and a tube-screen distance of 27 inches. Stage 2 plots the photon intensity after the beam's absorption by A, a typical adult anteroposterior chest projection; B, a typical adult anteroposterior abdomen projection and C, a typical adult lateral lumbar spine projection. Stage 3 is a plot of the photon intensity absorbed by the fluoroscopic screen; Stage 4, the light photon intensity emitted by the screen; Stage 5, the number of light photons per second emitted by 1 sq. mm. of screen surface and admitted by the pupil of the eye when the viewing distance is 20 cm.; and Stage 6, the number of these photons which, after admission to the eye, stimulate the retina.

It will be observed that the stage at which the number of photons per square millimeter per second reaches its lowest level is Stage 6, the stage at which photons are admitted to and stimulate the retina. When the data for Stage 6 are substituted in equation (1), the minimum perceptible images which can be seen at various levels of contrast for the three fluoroscopic projections under consideration may be easily calculated. The results of such calculations are shown in the dotted curves of Figure 7. The solid curves represent measured values of visual performance made on normal subjects. The ability of quantum statistics to describe the performance characteristics of the eye is apparent.

It will be observed from a perusal of Figure 6 that, if a screen intensifier with a photon intensification of 40 or more times were introduced between Stage 4 and Stage 5 of the fluoroscopic process, the stage at which the photon intensity reaches its lowest level would no longer be Stage 6 but instead would be that at which x-ray photons are absorbed by the fluoroscopic screen (*i.e.*, Stage 3). Under these circumstances, the quantum diagram for the anteroposterior chest projection would be that shown in the *solid* curve of Figure 8. Again,

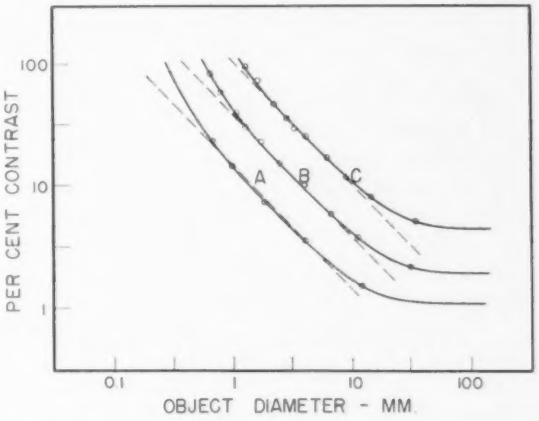

FIG. 7. Relationship existing between the diameter of the minimum perceptible image and image contrast during: A, anteroposterior chest fluoroscopy; B, anteroposterior abdominal fluoroscopy; and C, lateral lumbar spine fluoroscopy. Dotted curves represent values calculated from quantum statistics. Solid curves represent measured values of visual performance in normal subjects.

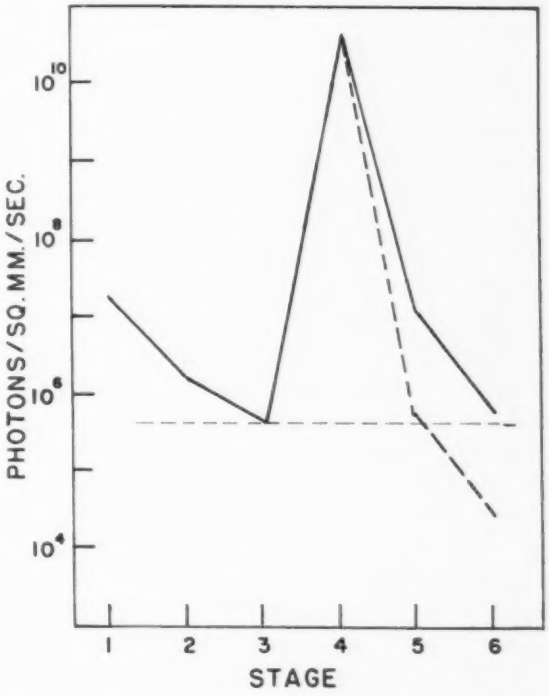

FIG. 8. Quantum diagram of the fluoroscopic processes when aided by screen intensification. Solid curve indicates data for a viewing distance equivalent to 20 cm. Dotted curve indicates data when viewing distance is equivalent to 100 cm.

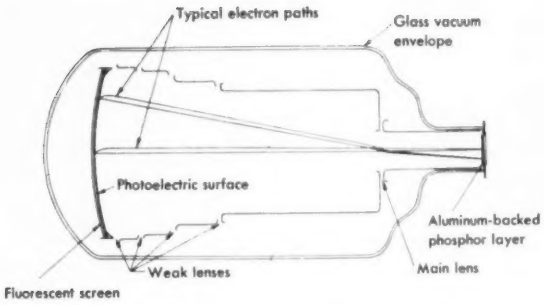

FIG. 9. X-ray screen intensifier of image amplifier type.

the data assume a viewing distance of 20 cm. It will be seen that, with the screen intensifier, the lowest level to which the quantum diagram descends is considerably higher than that when an intensifier is not employed. A considerable improvement may therefore be expected in the fineness of structure which may be perceived when such a device is used. Indeed, from Figure 8 and equation (1), the improvement should be of the order of 6 diameters.

IMAGE AMPLIFIERS

The most widely used type of screen intensifier is the electronic image tube or image amplifier,[1,3] an instrument which is shown schematically in Figure 9. This device consists of an evacuated glass envelope, at one end of which is an x-ray fluorescent screen located so as to intercept the x-ray photons emerging from the structure under examination; it is similar to the screens used in conventional fluoroscopy. A thin photoelectric layer is mounted on the screen's internal surface.

At the other end of the tube is a second fluorescent surface, whose diameter usually is only a small fraction of that of the first. This surface serves as a viewing screen and is backed with a thin aluminum layer to prevent its light from reaching the photoelectric surface at the other end of the tube. Between the two ends of the tube are placed a number of cylindrical electrodes which serve as electron lenses when suitable potentials are applied to them. Such an intensifier operates in the following manner: radiation from a conventional x-ray tube is projected through the structure under examination and is allowed to impinge on the tube's input fluorescent screen. In response to the light generated by this screen, the photoelectric layer emits electrons having a spatial distribution proportional to the brightness of the screen; that is, the fluorescent screen converts the x-ray image into a light image whereupon the photoelectric layer creates a corresponding electron image. Electrons from the photoelectric layer are accelerated through the length of the tube under the influence of the electrical potentials applied to the cylindrical electrodes and fall on the viewing screen at the tube's distant end. The impingement of electrons on this screen produces a visible image which duplicates the pattern of the x-ray fluorescent screen. However, due to the acceleration of the electrons within the tube, the brightness of the viewing screen is many times greater than that of the x-ray screen. The brightness is further enhanced by the fact that the size of the former is small compared to that of the latter. The observer views the intensified image by means of an eyepiece or other optical system to bring the image back to normal perspective. In most screen intensifiers of the electron image type, the intensification ranges from 500 to 3,000 times. Of this, however, from 30 to 100 times is the result of the minification of the image. Only 25 to 50 times is due to an actual increase in the number of light photons.

The improvement in fluoroscopic vision obtainable with intensifiers of the image amplifier type approaches the theoretic levels predicted by the data of Figure 8 if a viewing distance is used in which the visual field subtends the same angle to the eye as that when a conventional fluoroscope is viewed at a distance of 20 cm. Unfortunately, however, image amplifiers to be clinically practical require that the optical system place the viewer at an equivalent position substantially greater than 20 cm. from the screen. Indeed,

equivalent viewing distances in excess of 1 m. must be employed. Under these circumstances, the number of light photons admitted to the eye is substantially reduced; the performance of the intensifier is therefore degraded, and the quantum diagram of the fluoroscopic process assumes the pattern shown by the *dotted* curve in Figure 8. It will be seen that in this case the stage of the fluoroscopic process at which the number of photons per unit area per second reaches its lowest level is that at which photons are admitted to and stimulate the retina. Also, the quantitative level of this stage is not much greater than that of the similar stage in conventional fluoroscopy. Hence, image amplifiers when viewed with currently available optics do not yield a clarity which is much improved over that of conventional fluoroscopy. Of course there is a great advantage to screen intensification in its elimination of the need for dark adaptation. However, the full potential of screen intensification from the standpoint of its resolution or clarity has not been achieved.

Some improvement in the performance of image amplifiers has been made through their combination with television systems. With these systems the viewing conditions may be made sufficiently more satisfactory so that the resolution often is increased by a factor of 2. The contrast enhancement provided by television systems also improves the performance of screen intensifiers at low contrast levels.

SOLID STATE AMPLIFIERS

Although the image tube is, by all odds, the most widely used type of screen intensifier at the present time, several other varieties have been proposed. Some of these are not likely to find wide application, principally because they do little to increase the efficiency with which x-ray photons are utilized. One system,[2] however, which exhibits some promise is that shown in Figure 10. The system consists of two principal elements: a photoconductive layer which intercepts the x-ray photons coming from the structure under examination and an electroluminescent layer which emits light when it is placed in an alternating electric field. The two layers are separated by a thin film of opaque material of high electrical resistance. Also, a thin plate of conducting glass and a thin sheet of electrically conductive material such as aluminum are applied to the free surfaces of the electroluminescent and photoconductive layers, respectively. When the system is exposed to x-rays and a sinusoidal potential is applied to the aluminum and glass electrodes, a fluoroscent image of the structure under examination appears on the screen.

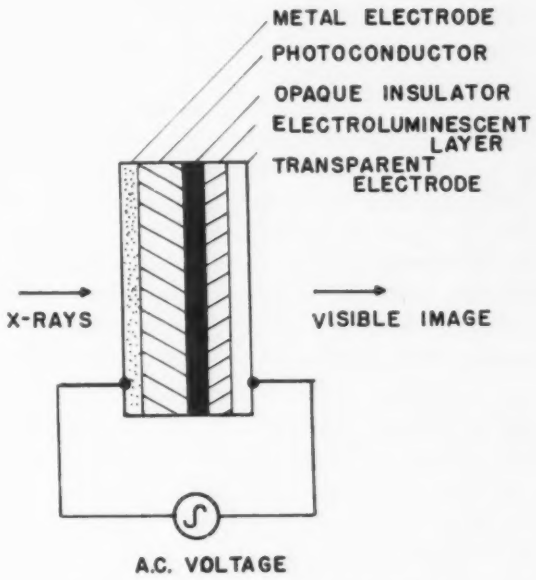

FIG. 10. Solid state screen intensifier.

The principles of operation are as follows: When x-rays are absorbed by the photoconductive layer, the layer's electrical resistance decreases and hence a greater fraction of the potential which prevails between the aluminum and glass electrodes appears across the electroluminescent layer. As a result, this layer emits light which may be seen by the observer through the glass electrode. Since the resistance of the photoconductive layer varies from place to place in accordance with the radiant intensity of the various x-ray images impressed

upon it, the electroluminescent layer will exhibit a series of images which conform to the x-ray pattern.

Many variations of the photoconductive, electroluminescent panel have been proposed and experimental models constructed. Although such panels are capable of producing light outputs which are several times those of conventional fluoroscopic screens, their response is unfortunately so slow that they are almost useless as fluoroscopic devices. Images smear badly when any motion is present. It appears that this characteristic is due to the rather poor mobility of the charges within the photoconductive and electroluminescent layers. Although it may be anticipated that this problem will eventually be overcome, this type of screen intensifier is not yet suitable for fluoroscopic work.

CINEFLUOROGRAPHY

As indicated earlier, screen intensifiers of the image amplifier type are proving of great value in cinefluorography. Indeed, without such instruments practical cinefluorography would not be possible because of the excessive radiation doses required when conventional fluoroscopic screens are photographed. The gains in illumination made possible by screen intensifiers have changed this situation entirely. In fact, when an electron image tube is used with a television system to increase the intensification still further, it is possible to make x-ray motion pictures with the use of no more radiation than that employed in fluoroscopy, even during the cinefluorography of dense abdominal structures.

In recent years, considerable controversy has arisen concerning the techniques best adapted for cinefluorography. Should one record directly from the image tube? Does recording from a television monitor provide the greatest number of advantages? Should one use 16 mm. film or 35 mm. film? There are many advocates of each technique, each group being certain that its method of operation is the preferable one. It is not surprising, then, that often those who plan to enter the field of cinefluorography become confused by the various claims and counterclaims. Unfortunately, most of the tenaciously held opinions are based upon limited experience where critical comparative experiments have not been carried out.

In spite of the confusion which exists within the field of cinefluorographic technology, experimental data are now at hand which substantially clarify the picture. These data show that, although the type of motion picture equipment employed in cinefluorography has some influence upon the clarity of reproduction, another factor is of more fundamental importance. This factor is the exposure dose rate applied to the patient during cinefluorography. Regardless of the equipment used, if the exposure dose rate is low, image quality will be poor. When the exposure dose rate is increased, image quality improves. The reason for this is well illustrated in the quantum diagram in Figure 11 where the photon

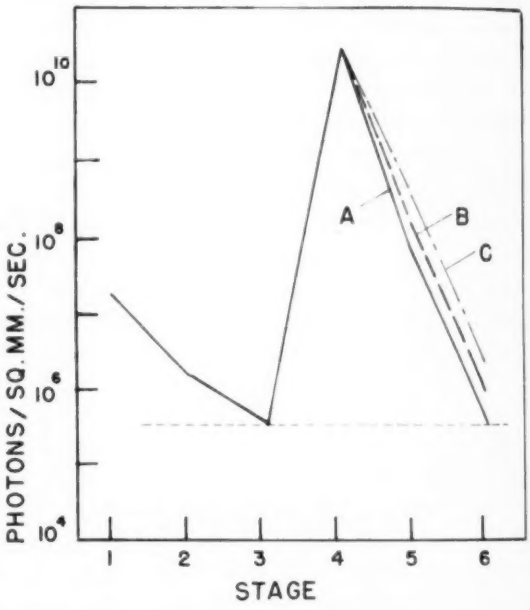

FIG. 11. Quantum diagram of the cinefluorographic process Data apply to a typical anteroposterior chest examination performed with a 9 inch image amplifier with an intensification of 3,000. Curve A, 16 mm. film and f/1.5 camera lens; curve B, 16 mm. film and f/1.0 lens; and curve C, 35 mm. film and f/1.5 lens.

intensity prevailing at the various stages of the cinefluorographic process is plotted for a typical anteroposterior chest cinefluorographic examination when a 9 inch image amplifier, having an intensification of 3,000, is employed. Stage 1 indicates the x-ray photon intensity (*i.e.*, the number of x-ray photons per square millimeter per second) prior to the absorption of the beam by the patient when the x-ray machine is operating at 90 kvp. and 10 ma. with 3 mm. aluminum filtration, a tube-screen distance of 27 inches and a cinefluorographic frame rate of 60 frames per second. Stage 2 indicates the photon intensity after absorption of the beam by the chest; Stage 3, the photon intensity absorbed by the input screen of the image amplifier; Stage 4, the number of light photons generated at the output screen by the photon intensity of Stage 3; Stage 5, the number of light photons admitted to the film by the optical system of the cinefluorographic camera by the photon intensity of Stage 3; and Stage 6, the number of photographic grains produced in the developed motion picture film by the photon intensity of Stage 3. Curve A represents conditions for 16 mm. cinefluorography in which an f/1.5 camera lens is employed. Curve B is for the same circumstances as Curve A except that an f/1.0 optical system is used. Curve C is for 35 mm. cinefluorography with an f/1.5 optical system.

It will be observed that in all cases, the stage at which the number of x-ray photons per unit area per second reaches its lowest level is the stage at which x-ray photons are absorbed by the intensifier (Stage 3) and not the stage at which x-ray images are recorded on motion picture film. Consequently, from quantum statistics, cinefluorographic detail under the conditions set forth in Figure 11 is not dependent on the type of film used but on the exposure dose rate prevailing at the input screen of the intensification system.

From a perusal of Figure 11, it will be apparent that, if the intensification of the image amplifier becomes less and/or the f/number of the camera lens is made greater, the performance of the cinefluorographic process ultimately deteriorates and becomes a function of the size of the recording film, since under these circumstances the stage of lowest photon intensity shifts to that at which film recording takes place. The deterioration first affects 16 mm. recording and for a given lens aperture is greater for 16 mm. film than for 35 mm. materials. However, as stated in foregoing paragraphs, when the lowest level of photon intensity occurs at the stage when the image amplifier's input screen absorbs x-rays, the type of film has very little to do with image quality. The circumstances under which this condition is satisfied is expressed by the formula:

$$\frac{TcaF}{4f^2m^2} \geq 1 \qquad (2)$$

where c is the number of light photons emitted by the input screen of the image amplifier per x-ray photon absorbed, a is the intensification factor of the image amplifier, T is the fractional transmission of the optical system, f is the effective f/number of the optical system, m is the minification of the cinefluorographic image in respect to the image appearing on the input phosphor of the amplifier, and F is the fractional efficiency of the photographic emulsion. Typical values for T, c, and F are 0.4, 2,000 and .005, respectively. The equation therefore reduces to:

$$\frac{a}{m^2f^2} \geq 1 \qquad (2a)$$

Values of m for 5 and 9 inch amplifiers and for 16 and 35 mm. film are given in Table 1.

Equation (2) is of fundamental importance in cinefluorographic design. It indicates when a 35 mm. camera is to be preferred to a 16 mm. unit; more important, it permits one to determine the conditions under which maximum image quality may be determined regardless of film size. For example, if one wishes to record from a 9 inch intensifier with a 16 mm. camera under

TABLE I

MINIFICATION VALUES FOR 16 MM. AND 35 MM. CINEFLUOROGRAPHY MADE FROM 5 and 9 INCH IMAGE AMPLIFIERS

Film	Image Amplifier	
	5 inch	9 inch
16 mm.	17	30
35 mm.	7	12.5

conditions when image quality will not deteriorate because of film size, equation (2) indicates that a lens having an aperture approaching f/1.0 must be used.

Equation (2) is also valid for cinefluorographic systems in which the recording is taken from a television monitor. Because of the higher intensification values these systems provide, one may employ relatively large f/numbers in the camera optics and still obtain maximal results with 16 mm. film. Indeed, the intensification factors are typically so large that 35 mm. film is quite unnecessary in this service.

The importance of the exposure dose rate prevailing at the input screen of the image amplifier in determining the quality of the cinefluorographic film is well shown in a practical manner in Figure 12. These films were made with a motion picture camera recording the information provided by an image amplifier equipped with an image orthicon television system. Frame A illustrates a view of the television monitor with an exposure dose rate at the input screen of the image intensifier of 500 μr per frame (the exposure dose per frame prevailing during cinefluorography of the chest with the motion picture camera operating at 60 frames per second and the x-ray equipment operating at 90 kvp. and 10 ma., with 3 mm. aluminum filter and a 27 inch

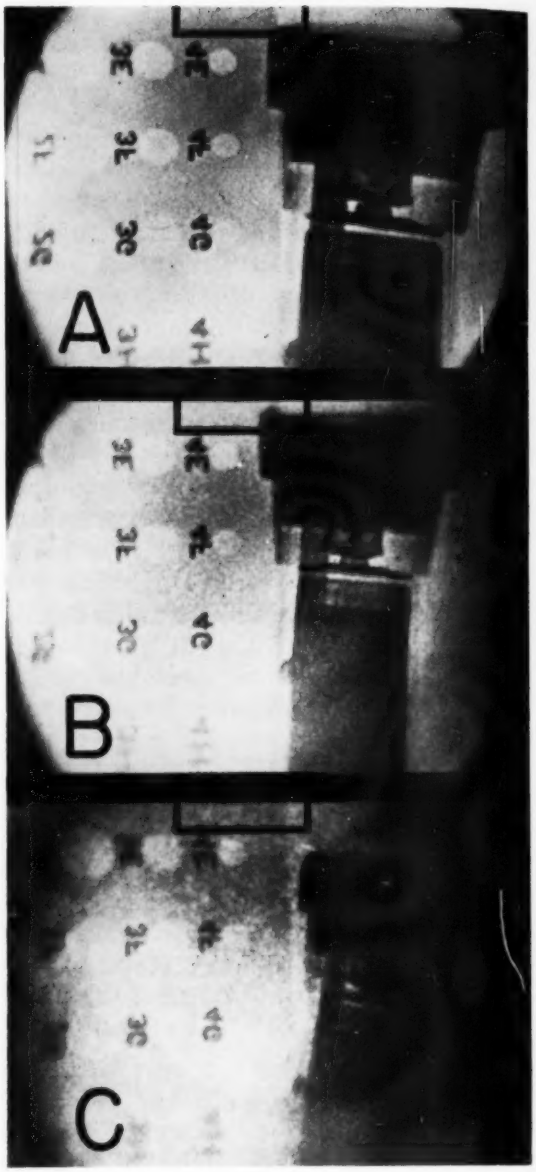

FIG. 12. Demonstration of the effect of exposure dose rate on cinefluorographic quality. Films were made from the television monitor of an intensifier consisting of a 9 inch image amplifier and an image orthicon television system. Frame A, single cinefluorogram with x-ray beam turned on at exposure dose rate of 500 μr per frame; frames B and C, same as frame A but with exposure dose rates of 50 and 5 μr per frame, respectively. Objects being roentgenographed were, on the left, a lucite plate with holes of varying size and contrast and, on the right, a flashlight. Note frank deterioration of image quality with decreasing exposure dose rate. Same cinefluorographic film, lens, and frame rate were used for all pictures. Compensation for de-

creasing screen brightness with decreasing exposure dose rate from frame to frame was provided by an increase in aperture of the lens between the image tube and image orthicon.

tube-intensifier distance). Frame B is the same as A but with the exposure dose rate per frame reduced to 50 μr. Frame C is the same as A but with an exposure dose rate per frame of only 5 μr. Although all films were made with the same motion picture film and with the television equipment performing under identical conditions, the influence of exposure dose rate on image quality is obvious. As the exposure dose rate decreases, the graininess visible in the picture becomes greater and the detail diminishes.

Since exposure dose rate values play a primary role in the determination of cinefluorographic quality, the question arises as to what cinefluorographic technique is best to employ under most circumstances. Usually, the convenience of 16 mm. film makes this material preferable to 35 mm. film, particularly if one has available intensification and optical systems adequate to make image quality independent of film size. In the case where motion picture films are recorded from a television monitor, there is no advantage in the larger film.

The choice between direct and television recording is more difficult. The maximum resolution of a 9 inch image intensifier is of the order of 0.25 mm.; that of an image amplifier equipped with an image orthicon television system is approximately 0.4 mm. Hence, direct recording produces somewhat greater detail at high exposure dose rates and with images of high contrast. Also, television recording is limited to frame rates of 30 frames per second and lower unless special equipment is used. Hence, this technique will usually not be suitable for many cardiac examinations where frame rates of 60 frames and over are desired. On the other hand, if a frame rate of 30 frames per second is adequate, television recording has many advantages. One of these is the increased intensification provided by television systems. As a result, abdominal cinefluorography of adults may be performed without excessive exposure dose rates. Another advantage is one's ability to change at will the intensification factor of a cinefluorographic system to meet the resolution requirements of the procedures to be recorded. The television method also has the advantage that it dissociates fluoroscopic and cinefluorographic operations. Hence, fluoroscopy can be conducted jointly with cinefluorography without one's having to discontinue one technique and start the other. This is particularly worthwhile in cinefluorography of rapidly changing events, such as those which occur in the pharynx during swallowing.

With the experience gained by many workers over the past few years, it is reasonably certain that cinefluography will play an ever increasing role in the field of diagnostic radiology. Even such techniques as myelography can be recorded by the cinefluorographic camera without excessive exposure of the patient. It is confidently anticipated that the next ten years will bring many advances in the diagnosis of disease by cinefluorography. Instruments capable of approaching the ideal in terms of performance are available. Their breadth of application only awaits the time needed to fully explore their potential.

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NORMAL ROENTGENOGRAPHIC ANATOMY OF THE HUMAN CIRCLE OF WILLIS*

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ROENTGENOGRAPHIC studies of the arterial system of the human brain are common. Methods of cerebral angiography in current use under normal circumstances opacify only isolated portions of the intracranial circulation. Usually, the posterior segment or the homolateral half of the anterior segment of the circle of Willis is visualized. Under these circumstances anatomic relationships of this important vascular complex must be inferred from a number of different angiographic studies. Such conditions make it exceedingly difficult to form valid estimates of normal anatomy in the circle of Willis or to judge the effects of health or disease upon it. Yet this arterial interchange is the structural distribution system which monitors regional cerebral arterial flow—and it is precisely in the field of disturbed *regional* flow that cerebrovascular disease exerts its effects. A reliable method of cerebral angiography which would produce simultaneous physiologic opacification of several or all anatomic parts of the circle of Willis might be expected to throw considerable light on normal hemodynamics in this little understood complex, and aid in evaluation of the effects upon cerebral circulation of disease or developmental deviation.

Once the feasibility had been established of routinely filling the drainage systems of carotid and vertebral arteries by retrograde brachial injection,[2] the submentovertical projection was added to the standard anteroposterior and lateral series for more complete angiographic survey of each patient. It was immediately discovered that in this view components of the circle of Willis in part, or in their entirety, were visualized with considerable clarity. The attainment of this objective has been reported elsewhere.[3] It is the purpose of the present communication to describe normal vascular patterns of the human circle of Willis which can be demonstrated following retrograde injection of the brachial artery, and to discuss certain functional implications that presented themselves during the course of these studies.

METHOD

Brachial cerebral angiography is best performed under local anesthesia.[4] Whereas percutaneous puncture techniques in the cervical region frequently produce unavoidable distress to patient and angiographer, brachial arteriotomy is easily accomplished in comfort. In the past, the problem posed by a frightened, struggling patient during percutaneous puncture of the carotid, vertebral or subclavian artery constituted a positive indication to some physicians for use of general anesthesia as a routine measure. Yet purposeful induction of unconsciousness in a patient with cerebral dysfunction of unknown cause is a hazard better avoided. Patients are much less averse to a small incision in the arm than to insertion of a needle in their neck. It is of interest that when brachial angiography is performed in a patient who had previously undergone percutaneous injection, reaction to the two procedures invariably is favorable to surgical approach in the arm.

A number of advantages is a consequence of the site of arterial cannulation. The patient is able to move about freely during the procedure; positioning of the head and neck with precision is a relatively simple maneuver; and injection of opaque medium produces discomfort mainly in the arm and shoulder where it is better tolerated than in the head. Still other important benefits include avoidance of

* This work was supported in part by a grant from the Tobacco Industry Research Committee.

trauma to major arteries of the cerebrovascular tree; circulation of opaque medium under conditions similar to physiologic flow of blood; and opacification of two-thirds or more of the cerebral circulation subsequent to each injection.

Retrograde brachial injection is carried out in a room especially equipped for such studies. Surgical technique is uncomplicated. The right upper limb is abducted in slight flexion at the elbow; surgical solutions are painted on the shaved upper inner arm; linear infiltration of a local anesthetic is carried out in skin overlying the bicipital groove; a 3 cm. incision is made, subcutaneous tissues are retracted and the neurovascular bundle is identified; then the brachial artery is isolated and lifted on rubber tapes. The artery is incised and a special twelve-gauge cannula* is inserted and anchored in place.

Positioning for the submentovertical view presents no difficulties. Usually these roentgenograms are obtained after completion of the anteroposterior and lateral sequences. The patient lies supine with his shoulders extending slightly beyond the head end of the stretcher-table. The latter is slowly pumped up until the vertex of the hyperextending head barely contacts the surface of the film-changer (Reed's base line parallel to the table). This position is surprisingly comfortable for most patients, and frequently easier to attain in the elderly than even moderate degrees of head flexion. The tube is adjusted so that the 10 cm. protective cylinder is inclined at an acute angle. The mandible of the patient is hyperextended to move it out of the way and the head is immobilized between plastic sponges. Ideally the mandible will then form the boundary along the upper edge of the exposed film sequence.

The time-setting sequence ordinarily is two films during the first second, then three films each second for the next several seconds. Delay roentgenograms are not obtained in the average patient since capillary and venous phases of cerebral circulation in submentovertical projection have not been found to be particularly informative.

* Manufactured by Becton-Dickinson Company, Inc., Rutherford, New Jersey.

DISCUSSION

Roentgenographic studies of the human circle of Willis have awaited a method which would provide successful opacification of anterior and posterior segments of this artery-complex while conditions of flow were maintained as nearly normal as possible. Retrograde cerebral angiography is considered by the author to fulfill for the human, the criteria laid down by McDonald and Potter[5] in their experimental investigation of cerebral circulation in the rabbit —avoidance of occlusion of the arterial lumen and lack of induced pressure changes in the system being investigated. After retrograde injection in the right brachial artery of man, the bolus of hypaque which reaches the subclavian-innominate lumina equilibrates with arterial pressure in a fraction of a second. Blood pressures measured on the opposite arm during and subsequent to injection have not been found to vary significantly. It is most likely, therefore, that following pressure equilibration in thoracic vessels the contrast medium is circulated through the carotid and vertebral systems under hemodynamic conditions normal for the individuals under investigation. Vascular pathways which become visible subsequent to injection in this fashion are believed to be those through which blood circulates in a physiologic setting.

It is not uncommon after retrograde right brachial injection to see in the initial high speed roentgenograms an advance column of diluted opaque medium streaming along the walls of carotid or vertebral arteries. The vessels have not yet been flooded with contrast solution. The central core of each artery remains for an instant translucent, testifying to higher speeds and greater dilutions in this region. During these transient phases, a hint can occasionally be gained of hemodynamics, for with incomplete opacification swirling and spiralling become visible, or laminar flow

at the head of the column can be seen to be interrupted by sudden turbulence. These features vanish with arrival of the main body of opaque medium, although they sometimes reappear intracranially when concentrated contrast solution in the vertebral artery meets a diluting stream at the basilar artery.

When the 50 per cent hypaque solution is undiluted by blood, limits of vessel size for visibility are surprisingly small. It is not infrequent that terminal branches of small arterioles (200–300 μ) are sharply opacified. Peculiarities of vessel structure, branching, and progressive filling are easily determined by examining consecutive roentgenograms, and artifacts due to positioning or confusing bone detail can usually be readily distinguished.

Filling and emptying of the human circle of Willis with contrast medium occur quite rapidly in the normal individual. A complete cycle through the anterior segment often takes place in less than two seconds. The speed of film changing is therefore greater for submentovertical projection so as to obtain a clear sequence of fluid flow through the system.

Velocity of blood flow is proportioned to the size of vessel through which it travels. It has been pointed out in a previous paper[3] that serial roentgenograms corroborate this relation between artery luminal diameter and speed of flow. Brachial-cerebral angiograms show a consistent difference in rate of flow through vertebral and internal carotid arteries. Normally, the rapidly moving stream of the internal carotid artery enters the cranium first, and opaque medium has moved well out into peripheral branches of the middle and anterior cerebral arteries by the time the advancing contrast medium-column in the more narrow vertebral artery arrives at the brain stem. This considerable time differential between anterior and posterior segment flow is maintained throughout the cycle of cerebral circulation and it is not uncommon to see opaque substance still within the basilar and posterior cerebral arteries during hemispheral venous phases.

Certain structural and functional components of the human circle of Willis are seen with regularity. There is little variability in the course or relative position of major trunks contributing to the circle, for example. But the origins and distributions of secondary arterial branches are subject to considerable individual difference, making it exceedingly difficult to distinguish normality from abnormality, or developmental difference from effects of vascular disease. Flow patterns of the circle of Willis will be described in patients who have shown no clinical manifestations of cerebrovascular disease. Deviations in structure or function felt to be significant will be commented upon.

Flow Patterns

A typical sequence of filling and emptying within the circle subsequent to retrograde injection of the right brachial artery is shown in Figure 1. Despite manual compression of the contralateral common carotid artery, crossover is not complete in the anterior segment, indicating lack of functional intercommunication between the internal carotid arteries. In the first film, hypaque in the carotid system has streamed several inches beyond the cervical bifurcation, while in the vertebral artery the head of the column has just come into view. One half second later (second film) peripheral branches of the middle and anterior cerebral arteries are well opacified, and contrast substance has reached the basilar artery. Subsequent exposures represent an elapsed time of one and one half seconds, during which fine radicles originating from the superior surface of the basilar artery become visible, and filling occurs in the large left posterior cerebral artery which remains a prominent vessel to the end.

As the basilar drainage opacifies, there can be seen small twigs representing the right posterior cerebral and spinal arteries, and a thread-like channel which is probably the left posterior communicating vessel. *Filling of the left posterior communicating artery represents the sole anastomotic response*

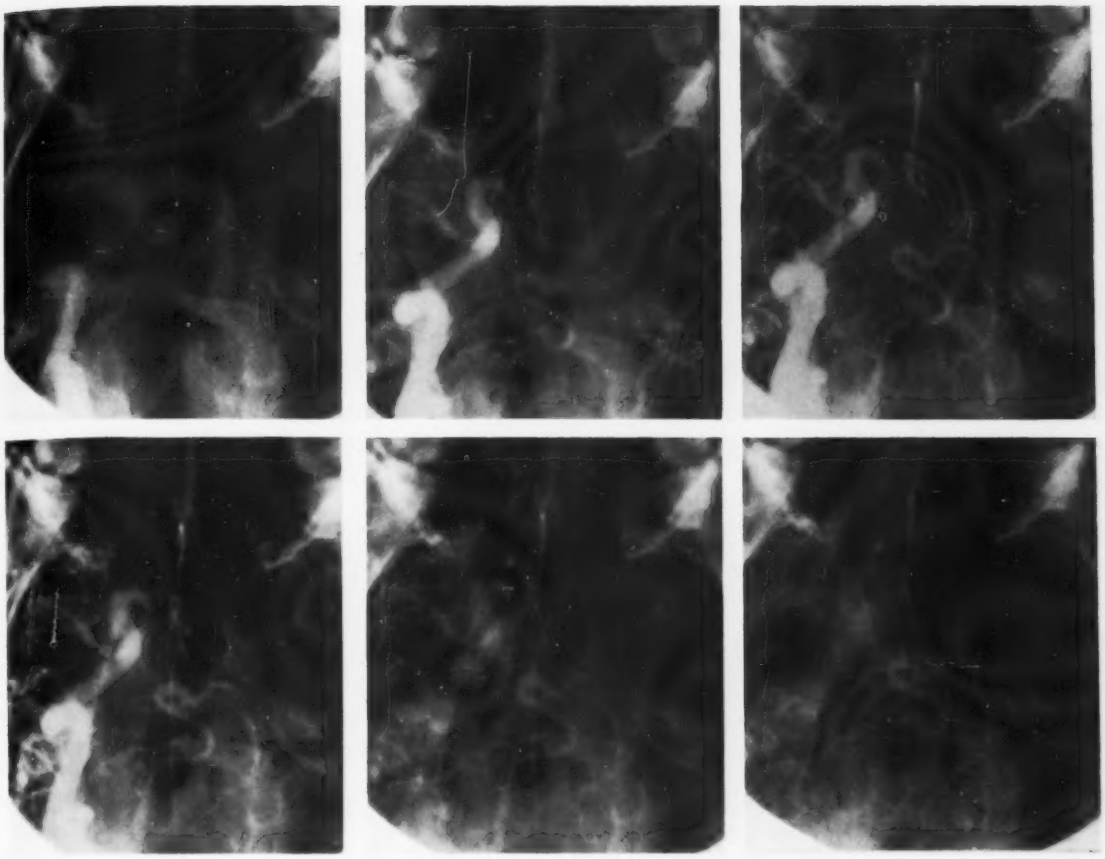

Fig. 1. Retrograde right brachial injection with contralateral carotid compression. Sequence begins at upper left and proceeds left to right. Circulation is normally rapid. Total time required is slightly over two seconds. The anterior segment does not communicate. Note the sharply curved course of the basilar artery and the characteristic persistence of the opaque medium in that circulation. The disproportion in diameter of the two posterior cerebral arteries is somewhat unusual. The anterior cerebral artery is superimposed upon the median nasal septum.

of this circle to marked drop of pressure in the contralateral carotid system. The anterior inferior cerebellar branch is very small. The diameters of vessels can be seen to remain constant throughout the circulatory cycle, and differences in size appear to be a reliable reflection of different hydraulic capacities in various branches of the system. It is noteworthy that the right posterior communicating artery is not visible during any part of the cycle, and also that both anterior cerebral arteries fill from the right side.

The large volume of opaque medium propelled up the carotid artery dissipates rapidly, and comparatively little remains in this system on the final film. Speed of flow in the basilar system is in marked contrast, for opaque substance persists until the end of the film sequence. The total time that contrast medium remains within the anterior segment of the circle is much less than the period for which it lingers in the posterior segment.

In all sequences to date it has been observed that, unless there is arterial obstruction or major structural deviation in the carotid system, posterior segment flow is delayed and relatively sluggish. It has, therefore, been assumed that this is a natural phenomenon. The significance of normally more rapid circulation through drainage fields of the anterior and middle cerebral arteries is as yet unknown.

Examples of different arterial configura-

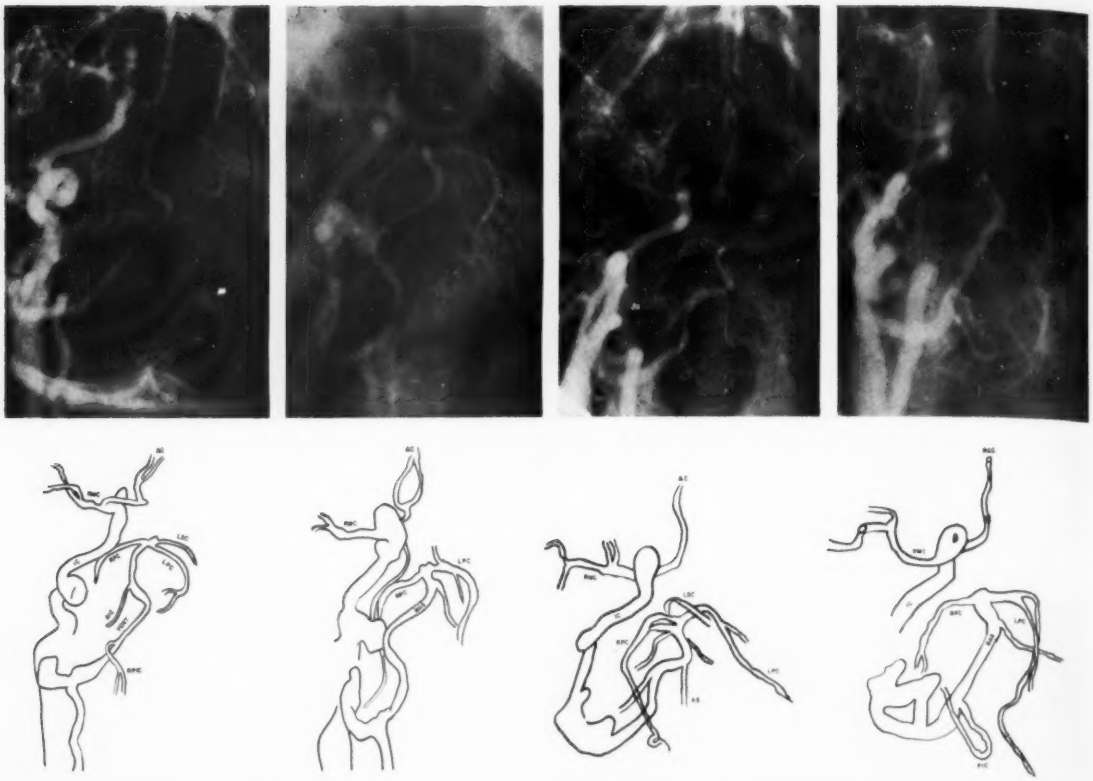

FIG. 2. Configurations of the circle of Willis. Each tracing is of the roentgenogram above. No carotid compression was performed. Note the consistent bilateral filling of the posterior cerebral arteries and the bilateral opacification of the basilar branches. A posterior communicating artery (left) is seen on only one roentgenogram. There is great variability in the site of origin and the course of vessels derived from the basilar artery.

tions of the human circle of Willis during maximal filling are shown in Figures 2 and 3. These patterns are alike in that fluid flow in the anterior segment of the circle remains unilateral despite efforts in some cases to produce anastomotic communication by contralateral carotid compression.

Carotid Trunks. The common carotid artery is seen as a heavy, opaque column extending several inches from the lower margin of exposed film. A fairly abrupt, well-marked narrowing marks the point of cervical carotid bifurcation. The internal carotid artery then ascends vertically a short distance, swings sharply to pass medially in a straight line for about 3 cm., and reverses course to penetrate the floor of the skull. Variations are unusual in the cervical course of these major trunks unless arterial disease is present.

Anterior Cerebral Artery. Opacification of the right anterior cerebral artery is such a constant feature of retrograde angiography that nonfilling is regarded as evidence of occlusive vascular disease or major developmental deviation. The precise incidence is unknown of derivation of both anterior cerebral arteries from one internal carotid, but it must be quite low.[6] In the present series of approximately 50 consecutive cases, including patients with various kinds of cerebral lesions, only two examples were found of dual unilateral derivation (Fig. 1). Multiple channels connecting anterior cerebral arteries are known to be far more common than inadequate communication between these vessels—a situation quite unlike that characteristic of posterior communicating vessels.[1]

During early filling phases the anterior

cerebral arteries overlie and parallel the median nasal septum, whereas a second or so later their terminal branches arch backward and may be superimposed on the circle of Willis itself. Moderately high-speed film shuttling (3/second) allows easy differentiation of these branches from those arising at the circle of Willis.

Posterior Segment Arteries. The vertebral artery displays little variation in its course, and the characteristic cervical loop[4] is well displayed in submentovertical views. A branch of this artery, visible on most angiograms, is the right posterior inferior cerebellar artery (Fig. 2 through 5, inclusive). This vessel arises from the medially ascending limb of the vertebral artery, and follows a highly variable path. On an occasional angiogram a parent trunk of the anterior spinal artery can be seen arising in approximately the same location.

The Basilar Artery. The lumen of the basilar artery normally is completely filled during right brachial angiography. A curving course, sometimes C-shaped and occasionally in the form of an S, is much more characteristic of this vessel than the straight median structure seen in textbook diagrams of the human circle of Willis. Although in some instances of unusual tortuosity, atherosclerosis is undoubtedly a contributing factor, it has been observed that even in young persons free of serious arterial disease the trunk of the basilar artery may be sharply curved to the right or left. The rostral tip of the artery, on the

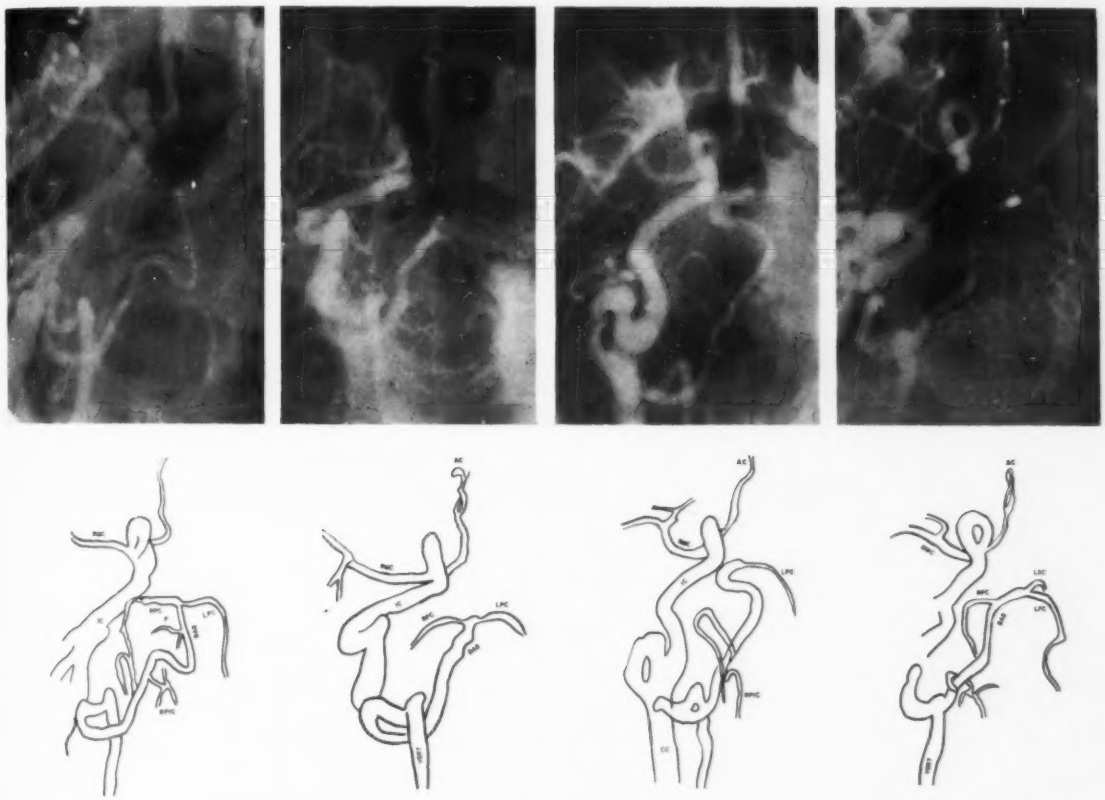

Fig. 3. Configurations of the circle of Willis. Each tracing is of the roentgenogram above. Carotid compression carried out during injection of patient on far right results only in slight reflux into left anterior cerebral artery. Note the wide differences in diameter and variable course of the basilar artery. The right posterior cerebral artery fails to fill in one instance (? pathologic change). The middle and anterior cerebral arteries show little variability in origin or distribution of their main trunks. The posterior communicating arteries are usually not seen.

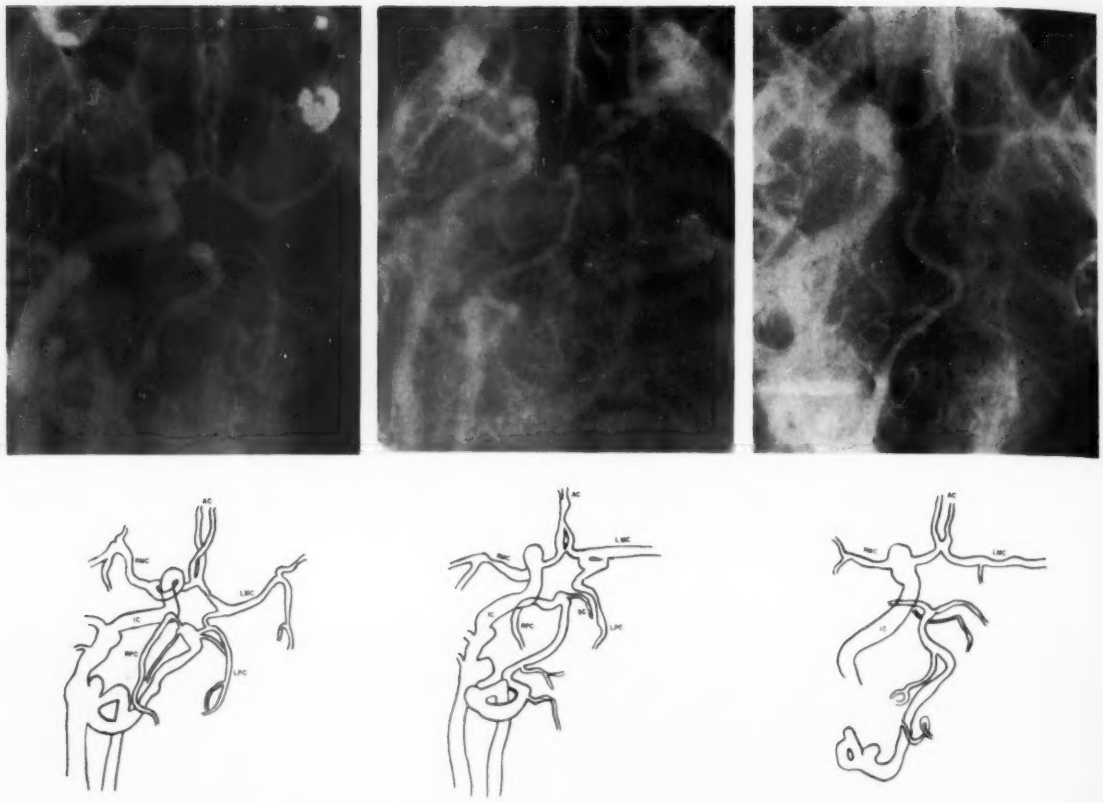

FIG. 4. Brachial retrograde injection with compression of contralateral cervical carotid artery. Filling of the entire anterior segment of the circle of Willis is demonstrated. *Left:* The slender right posterior communicating artery is well seen. Note the symmetry of the two middle cerebral arteries and the apparent transposition of the anterior cerebral arteries. The vertebral artery is of adequate size and the superior cerebellar branches are filled. *Middle:* Note the derivation of the left posterior cerebral artery from the internal carotid artery and the median but narrow basilar artery. *Right:* C-shaped basilar artery with a prominent right posterior cerebral branch.

other hand, varies little in its central anatomic position from patient to patient.

Posterior Cerebral Arteries. Both posterior cerebral arteries can be clearly seen in the usual study. They arise almost always from the rostral end of the basilar artery and appear to be a natural continuation of this vessel. Density of opaque medium is the same in each of the vessels. This bilateral circulation is in marked contrast to that described for animals,[5] and indicates that thorough mixing of the vertebral streams is the rule in the human basilar artery. It is probable that interruption of laminar flow by turbulence at the vertebral-basilar junction is responsible for the phenomenon. The two posterior cerebral arteries demonstrate typically a "weeping-willow" configuration. Major variations are common, and may include bifid origin, early branching, and occasionally what appears to be total absence of one artery (Fig. 2).

Other Basilar Branches. Superior cerebellar arteries are seen with a fair degree of frequency. It is only rarely possible to identify a sequence of lateral branches of the basilar artery similar to those ordinarily diagrammed in texts. This discrepancy cannot be ascribed to physical characteristics of the opaque medium used for angiography, for left-sided basilar branches fill almost as frequently as those on the right, and intracranial arteries of very small

caliber regularly become opacified elsewhere (Fig. 3). As a matter of fact, it is not uncommon during certain arterial filling phases to see fine perforating radicles emerging from the superior surface of the basilar artery, or hair-like pontine branches outlining the external form of the brain stem.

When contralateral carotid compression is carried out, a few seconds before retrograde brachial injection, opaque medium may cross to fill the entire anterior segment of the circle of Willis. This has occurred in perhaps 50 per cent of individuals to date. The distal internal carotids and right and left anterior and middle cerebral arteries are flooded with contrast solution. Shortly thereafter the posterior segment fills via the vertebral artery and all major functional components of the circle can be visualized. Figures 4 to 6, inclusive, show the roentgenographic appearance of the circle of Willis at that instant. Occasionally, similar filling will occur when an excess of opaque medium refluxes proximal to the innominate into the arch of the aorta and passes up the left internal carotid artery (Fig. 6).

Filling of the entire anterior segment of the circle of Willis is excellent evidence of anastomotic response to drop of arterial pressure in the left carotid system, and proof of the existence of adequately patent anterior communicating channels. The more rapid anterior segment circulation results in filling of the entire system bilaterally, before the basilar drainage becomes opacified by arrival of the vertebral stream. There is no evidence that opaque medium

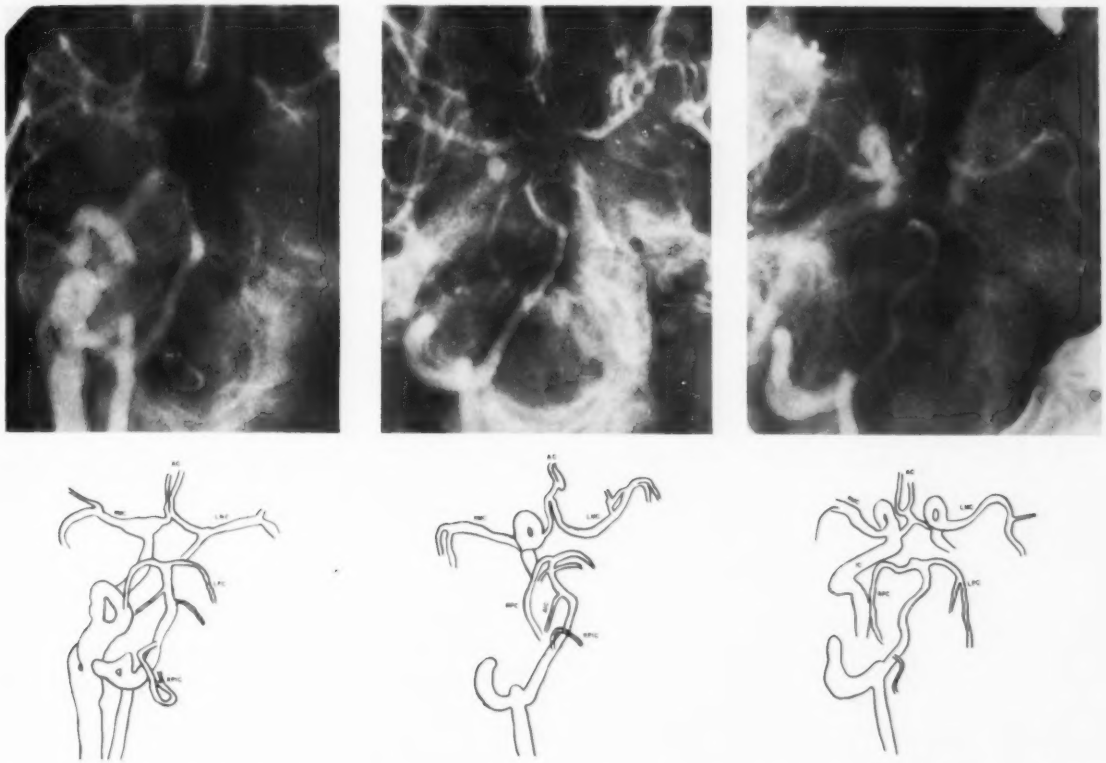

Fig. 5. Right brachial injection and left carotid compression. *Left:* Quadrilateral circle of Willis with unusually thick left posterior communicating artery joining the posterior cerebral trunk. Note the prominent posterior inferior cerebellar branch and the midline basilar artery. *Middle:* Posterior dislocation of the right middle cerebral complex by space-occupying mass. The posterior cerebral and superior cerebellar branches are well outlined. *Right:* Symmetric anterior and middle cerebral arteries with widely-spaced "weeping-willow" posterior cerebral configuration. There are no visible posterior communicating arteries.

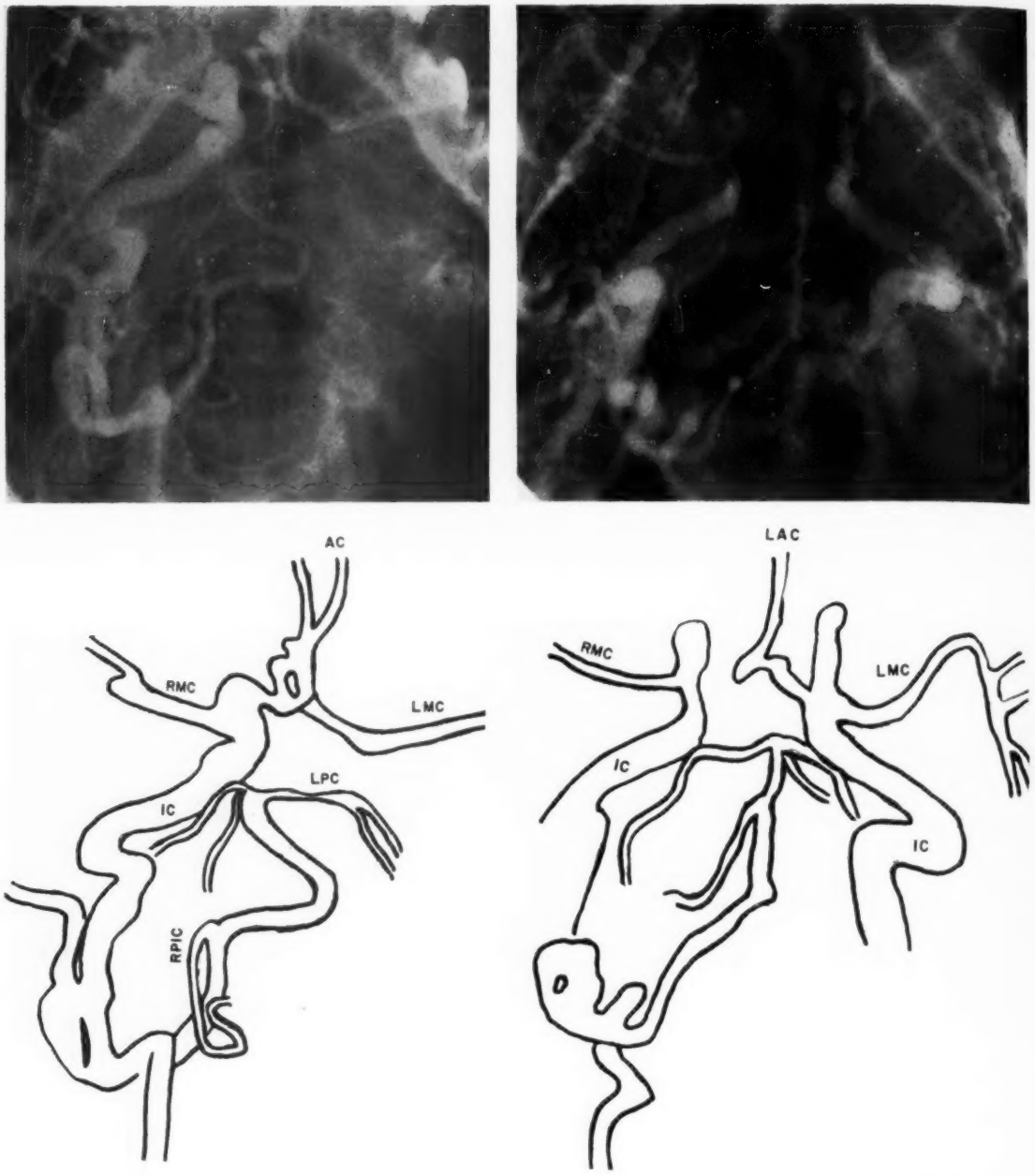

Fig. 6. Brachial injection with left carotid compression. *Left:* Prominent posterior inferior cerebellar artery and almost transversely placed basilar artery are visualized. *Right:* The opaque medium ascends both internal carotid arteries. Filling of the anterior cerebral arteries is seen only from the left. Note the markedly different course of the right and left middle cerebral arteries. There was no clinical evidence suggesting the presence of an intracranial lesion.

flooding the anterior segment under these conditions passes posteriorly to contribute to the basilar circulation nor that circulation in the basilar system compensates for the pressure drop in the left anterior segment. During this type of maximal circulatory stress, therefore, the left posterior communicating artery, at least, fails to respond as an anastomotic conduit. The posterior communicating vessels can rarely

be seen during brachial-cerebral angiography and, in the few instances they are visible, they are of such small caliber that it is difficult to conceive them to be carrying significant volumes of blood.

These observations indicate that the human circle of Willis is functionally a circle in name only. The independent anterior and posterior segments possess major differences in function. Internal carotid flow normally supplies the ipsilateral half of the anterior segment, but about 50 per cent of individuals possess the capacity to maintain adequate functional circulatory flow to both hemispheres by way of one internal carotid artery.

Posterior segment circulation is characterized by bilateral distribution of a relatively small volume of slowly moving arterial blood. The brain stem and cerebellar areas are, therefore, usually isolated from the rapidly moving high volume stream perfusing the anterior segment. In the usual individual if the internal carotid supply fails bilaterally there would appear to be little chance that hemispheral perfusion could be supported by way of posterior segment flow.

SUMMARY

1. Retrograde brachial angiography fulfills experimental criteria established previously in animals for investigation of cerebral circulation. The technique avoids completely pressure artifacts and arterial injury.

2. When the submentovertical projection is combined with brachial injection, clear visualization is obtained of components of the circle of Willis.

3. Typical configurations of human circles are presented, and the influence of contralateral carotid compression is demonstrated.

4. Some preliminary observations are made on functional capacity of the human circle of Willis as demonstrated by this method.

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VALVES OF THE COMMON CAROTID ARTERY DURING ANGIOGRAPHY*

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THE common complications of carotid angiography have been reported in detail and are well known.[1] However, there is one uncommon complication which we feel has not received enough attention—the creation of artificial valves in the common carotid artery during the puncture of this vessel. Sirois, Lapointe, and Côté[6] have published such a case with postmortem findings. The purpose of this paper is to present 4 similar cases and to describe the roentgen findings.

All examinations were performed with a serial cassette changer exposing six films in sequence at one second intervals. The first 3 cases were studied by the percutaneous method while in the fourth case the common carotid was surgically exposed.

REPORT OF CASES

CASE 1. J.G. KCH #43553. This twenty-three year old white female entered the hospital on September 19, 1958 because of vaginal bleeding. The patient had had her last menstrual period three months before. According to the history she had suffered a generalized convulsion of five to ten minutes' duration a few hours before admission. There were no sequelae. A dilatation and curettage were performed on the following day. Six hours after operation the patient had a generalized convulsion following which she was unresponsive for a period of three days. Lumbar puncture performed during this period revealed xanthochromic spinal fluid. She subsequently became alert and there were no neurologic signs except nuchal rigidity.

Left carotid angiography was performed on September 30, 1958. No contrast material was seen intracranially until the five second angiogram. It was therefore suspected that there was a partial block in the cervical portion of the internal carotid artery. The films were then positioned for study of the neck and the examination was repeated. On this study the early angiograms revealed the contrast material to stop abruptly in the distal portion of the common carotid artery (Fig. 1). The head of the contrast column had a meniscus appearance with concavity directed inferiorly. On the three and four second angiograms the vertebral artery was faintly opacified. At five seconds (Fig. 2) the contrast material started to escape the block and eventually opacified the internal, external, and branches of the middle cerebral arteries. However, most of the contrast material remained in the distal portion of the common carotid artery and maintained the meniscus appearance on its upper border. A repeat examination performed immediately revealed the same findings. By October 5, 1958 the patient's nuchal rigidity had disappeared and two days later a lumbar puncture revealed clear spinal

FIG. 1. Case I. Three second angiogram showing contrast material in the common carotid artery.

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fluid with a negative Pandy test. The patient signed her release against advice on October 12, 1958. The discharge diagnosis was subarachnoid hemorrhage of undetermined etiology.

Case II. H.B.N. KCH #49727. This forty-two year old Negro male was admitted because of right hemiplegia and aphasia. While being admitted he developed a grand mal type of convulsion with loss of sphincter tone and went into status epilepticus for about thirty minutes. Toward the end of the convulsive episode the patient was noted to have more prominent convulsive movements on the right side of the body. Following this he was lethargic and pain stimuli caused motion of the extremities on the left side only.

He slowly became fully alert and seventeen days later, on November 6, 1959, left carotid angiography was performed. The operator had difficulty in puncturing the common carotid artery and the first injection was into the soft tissues of the neck. On the second injection it was noted that the needle was low in the common carotid artery and there were several filling defects in the opaque column just above its insertion. The contrast material passed these defects readily but stopped abruptly just below the common carotid bifurcation (Fig. 3). On the five second angiogram the carotid bifurcation was faintly outlined and at six seconds (Fig. 4) it was well outlined. On both these angiograms there was a faint line of radiolucency running across the opaque column just below the common carotid bifurcation. This line of radiolucency corresponded to the area where the opaque column had stopped on the early angiograms. In the anteroposterior projection it was noted that the common carotid artery appeared to be ballooned just below its bifurcation. Retinal artery pressures recorded on November 9, 1959 were 147/67 gm. in the right eye and 144/60 gm. in the left eye.

Fig. 3. Case II. Three second angiogram showing numerous filling defects in the common carotid artery. The opaque column has a rounded upper border.

Fig. 2. Case I. Five second angiogram showing some contrast material escaping the block in the common carotid artery.

The left carotid angiography was repeated four days later. At this time there was a smooth, rounded, elongated filling defect on the posterior aspect of the opaque column in the common carotid artery. This was apparently caused by a hematoma from the previous examination. Whether it was retrocarotid or intramural could not be determined. Above the defect a curved line of radiolucency arose from the posterior surface of the common carotid artery at the bifurcation and extended anteriorly and inferi-

Fig. 4. Case II. Six second angiogram showing opacification of the carotid bifurcation. There is a faint line of radiolucency just below the bifurcation.

orly to become lost in the defect. There was no obstruction to the flow of the contrast material and a normal cerebral angiogram was demonstrated. The retinal artery pressures measured on November 16, 1959 were equal. A pneumoencephalogram on November 18, 1959 was normal.

Left carotid angiography was again performed on December 14, 1959 (Fig. 5). At this time the previously described filling defect had become slightly flattened but was still quite apparent. It was now felt that this was an intramural hematoma which was undergoing organization. The curved line of radiolucency could still be seen, although it was not as prominent as on the previous study. The patient was eventually discharged with the diagnosis of a convulsive disorder of unknown etiology.

Case III. C.C. KCH #5056. This fifty-one year old Negro male was a chronic alcoholic and had been drinking heavily on the night prior to admission. He was found lying in bed on the day of admission in a stuporous condition. Information from a friend disclosed that the patient had been suffering from "circulation" in the legs recently. Upon admission he was stuporous and in shock. There was spasticity of both lower extremities, somewhat more marked on the left. There was a positive left Babinski toe sign. During the next two days the spasticity of the lower extremities decreased markedly and the Babinski toe sign disappeared. The patient remained stuporous. Lumbar puncture on the third hospital day (February 3, 1960) revealed clear fluid with a negative Pandy test and no cells.

Right carotid angiography was performed on February 4, 1960. Angiograms taken in the anteroposterior projection revealed the contrast material to stop abruptly in the distal portion of the common carotid artery (Fig. 6). The head of the opaque column had the appearance of a meniscus with its concavity directed inferiorly. In addition there was a faint crescentic line of radiolucency about 1 cm. below the point of the needle. In the early angiograms the contrast material flowed in a retrograde direction to opacify the right subclavian, innominate, and right vertebral arteries. In the four second and five second angiograms (Fig. 7) the contrast material slowly escaped the block and outlined an area of irregularity in the proximal portion of the internal carotid artery. A second needle was then inserted higher up in the common

Fig. 5. Case II. Angiogram made five weeks after Figures 3 and 4. The valve can be faintly seen. The smooth filling defect on the posterior aspect of the artery is apparent.

carotid artery and a repeat study was performed (Fig. 8). This showed an apparently normal carotid bifurcation and normal internal and external carotid arteries. Left carotid angiography was then performed and was negative. Both anterior cerebral arteries filled and were midline. There was no opacification of the right internal carotid bifurcation or right middle cerebral artery.

The patient was taken to the operating suite a few hours later and under local anesthesia the right common carotid, the bifurcation, and internal and external carotid arteries were exposed. Inspection showed that there were adequate pulsations in these vessels. A linear incision was made in the anterior aspect of the common carotid artery, the bifurcation, and the proximal segment of the internal carotid artery. There was a fresh intramural blood clot dissecting between the intima and media of the posterior wall measuring about 1½ cm. long, 4 mm. wide and 3 mm. thick. The clot was removed without difficulty. The artery was closed in two layers, intima to intima and media to media. Following the removal of clamps there were good pulsations in all the exposed arteries. Postoperatively the patient remained stuporous for twenty-one days. When he finally regained consciousness his sensorium remained clouded and he had a spastic quadriplegia.

Fig. 6. Case III. Early angiogram showing abrupt stoppage of the contrast column below the carotid bifurcation and retrograde flow through the subclavian, innominate and vertebral arteries. Note the curvilinear line of radiolucency just below the point of insertion of the needle.

Fig. 7. Case III. Angiogram shortly after Figure 6 showing contrast material outlining a filling defect above the point of previous blockage.

The right carotid angiography was repeated on April 27, 1960 (Fig. 9). This study showed a small area of irregularity in the proximal portion of the internal carotid artery which presumably represented postoperative changes. In addition, on the lateral view there was a line of radiolucency lying just anterior to the posterior border of the distal portion of the common carotid artery. The patient's condition did not improve and he was eventually transferred to a State hospital.

Fig. 8. Case III. A second injection has been made above the block seen in Figure 6 and a normal bifurcation is demonstrated.

Case IV. F.T. This fifty-two year old white male had been suffering from mild attacks of coronary insufficiency for the past ten years. He had had five hospital admissions during the period from October, 1956 to November, 1959, always with the chief complaint of sudden onset of unconsciousness not connected with any convulsive movements, and always with a negative neurologic examination. Because of the possibility of a cerebral vascular insufficiency syndrome, bilateral carotid angiography was performed in November, 1959 using the open technique.

Examination of the early right carotid angiograms revealed the contrast material to stop abruptly in the common carotid artery. The upper border of the opaque column had the appearance of a meniscus with the concavity directed inferiorly. On the three second and four second angiograms the vertebral artery was opacified. On the five (Fig. 10) and six second angiograms a small amount of the contrast material was seen to have escaped the block. However, most of the contrast material remained proximal to it.

Fig. 9. Case III. Angiogram made eleven weeks after Figures 6, 7, and 8. A valve can be seen in the upper portion of the common carotid artery and an operative defect in the proximal portion of the internal carotid artery.

The patient was later taken to the operating suite and the right common carotid artery and

Fig. 10. Case IV. Five second angiogram showing that most of the contrast material is in the common carotid artery and that the column has a rounded upper border. Some opaque medium is escaping the block to faintly outline the carotid bifurcation.

its branches were exposed. There were numerous adhesions behind the common carotid which were considered to be pathologic. There was an oval circumscribed bluish area approximately 2×1 cm. in the medial wall of the vessel about 3 cm. below the bifurcation. This was believed to represent a focal infarct of rather recent origin. In addition there was a ridge of scar tissue on the anterior aspect of the common carotid about 2 cm. below the area of infarction. There was no evidence of atheromatous change at palpation and powerful pulsations were felt in the common carotid, internal carotid and external carotid arteries. It was also noted that the distal portion of the common carotid artery just below the bifurcation was approximately twice the diameter of the more proximal portion of this artery. No attempt was made to open the carotid vessels.

DISCUSSION

We agree with Sirois *et al.*[6] that the angiographic appearance in these cases is caused by the formation of a valve. The point of the needle first pierces the intima on the posterior wall of the common carotid artery (Fig. 11). This is followed by a lateral movement of the needle slicing the intima in a crosswise direction. The pressure of the blood flow then elevates the intima and strips it from the media to form a valve. The tip of the needle thus comes to lie inferior and posterior to the lip of the valve. At this point the valve does not entirely occlude the lumen of the artery (Fig. 12). When the contrast material is forcefully injected, the valve is elevated further, coming into contact with the anterior wall of the artery and completely occluding the vessel. During this period the artery may appear to be ballooned just below the block and contrast material may opacify the vertebral artery by retrograde flow. The outline of the head of the contrast material in the common carotid artery is that of a meniscus with the concavity directed inferiorly. This condition lasts for three or four seconds and then the contrast material starts to escape around the valve as the pressure of the injection diminishes and the pressure in the common carotid artery returns to normal. We then see faint opacification of the bifurcation and proximal portions of the internal and external carotid arteries. However, most of the contrast material remains trapped proximal to the valve.

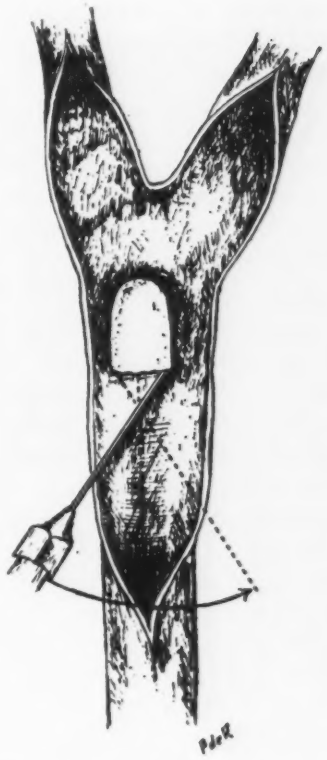

Fig. 11. Diagram showing how the intima of the posterior wall of the common carotid artery is sliced in a crosswise direction. (Reproduced with permission of the authors and *Journal of Neurosurgery*.[6])

The faint crescentic line of radiolucency seen below the point of the needle in our

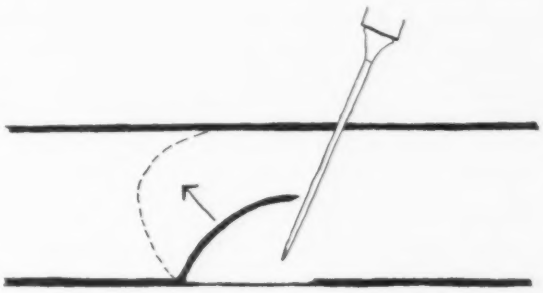

Fig. 12. Line drawing showing the valve in profile. The dotted line represents the position of the valve during and immediately after injection.

third case has also been described by Sirois and co-workers.[6] It is apparently the edge of the elevated intima. This finding cannot always be distinguished on the angiograms. The ballooning of the distal portion of the common carotid artery is likewise not always present. This latter sign may depend on the direction in which the opening of the needle is facing. If it is directed superiorly the contrast material will be forceably injected into the blind sac formed by the valve, thus causing expansion of the arterial wall. If it is facing inferiorly the force of the injection will be dissipated to a great extent against the blood flow and the arterial wall may not dilate.

The most reliable signs of the presence of a valve are the meniscus appearance of the upper border of the contrast column and the eventual escape of a small amount of the material, revealing a normal bifurcation, while the major portion remains proximal to the area of obstruction.

Review of 1,224 angiograms in our series has revealed only 3 cases of valve formation, giving an incidence of slightly less than 0.25 per cent. Because adverse signs and symptoms are rare following carotid artery puncture, we are probably unaware of the actual incidence of intimal damage without valve formation as a result of the procedure. This may be much higher than is suspected. Rowbotham *et al.*[5] performed postmortem examinations on 6 patients within ten days after they had undergone carotid angiography. In all cases the intima on the posterior wall of the carotid vessels had received one or more lacerations up to 3 mm. in length. In one instance there was a hemorrhage in the media 1 cm. long and 5 mm. across. This would correspond to our Case II and Case III. In another of their cases, there was a friable thrombus projecting into the lumen of the vessel from one of these lacerations.

If a valve of the common carotid artery is suspected, a second needle should be inserted above the involved area into the common or internal carotid artery. This was done in our third case but we failed to realize the significance of a normal carotid bifurcation. The measurement of retinal artery pressures may also suggest the presence of this condition. If there is not more than a 15 per cent difference in the systolic retinal artery pressures, occlusion of the common carotid artery is unlikely.[4] Our Case II should have indicated this to us. Pressure on the contralateral carotid artery, as advocated by Gurdjian and co-workers,[2,3] may also be of help in the detection of this condition.

SUMMARY

1. Four cases of artificial valves of the common carotid artery are presented.

2. An artificial valve is formed during carotid angiography by the needle piercing the intima of the posterior wall of the common carotid artery and moving in a crosswise direction. The intima is then elevated and stripped from the media by the pressure of the blood flow, thus forming a valve. This valve causes a partial obstruction to blood flow under normal conditions and a complete obstruction under the abnormal conditions produced when the contrast material is injected.

3. If a valve is suspected, a second needle may be inserted higher up in the common carotid artery or in the internal carotid artery. Measurement of the retinal artery pressures and compression of the contralateral common carotid artery may also help to rule out an occlusion.

4. This lesion is important not only because it may lead to the erroneous diagnosis of an occlusion of the common carotid artery but also because of its possible sequelae, namely, dissecting hematoma and thrombus formation.

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The author wishes to thank Dr. John P. Gallagher of Washington, D.C. for his kind permission to publish Case IV.

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THE ROENTGEN IMAGE OF THE SUBCLAVIAN ARTERY IN THE PULMONARY APEX

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THE subclavian artery may cast a shadow in either pulmonary apex and can be recognized as such on a roentgenogram of the chest. In a review of 300 normal chest roentgenograms it was seen in one apex or in both apices in 40 per cent of the cases. It was more commonly observed on the left than on the right side, the approximate incidence being 2:1. It appears as a curved homogeneous shadow 3–4 cm. in length, sometimes sharply but more often indefinitely delineated (Fig. 1, *A* and *B*). It is projected in one of the four proximal posterior intercostal spaces, most commonly in the second and third; however, it may be completely or partly obscured by the ribs. When it is distinctly outlined, it can be followed on the left as a continuation of the ascending portion of the subclavian artery. This portion is commonly seen, together with the common carotid artery, as a homogeneous shadow just to the left of the air column in the trachea. On the right, the subclavian artery may be identified as a branch of the innominate artery.

REVIEW OF THE LITERATURE

In a contrast study on a cadaver, Assmann[2] has demonstrated that the shadow described above is that of the subclavian artery. Since the advent of angiographic contrast studies *in vivo*, the location and the appearance of the opacified subclavian artery on chest roentgenograms has become well known. A well-documented paper on this subject was presented by Poker and associates.[8] Nevertheless, the identification of the subclavian artery with the shadow seen on plain chest roentgenograms was not made. Poker and associates state that "Roentgenographically, the subclavian arteries are invisible in frontal and lateral views. However, the left subclavian artery can be visualized in the left anterior oblique view. . . ."[8] The last sentence of the quoted statement refers to the first portion of the artery, which represents the

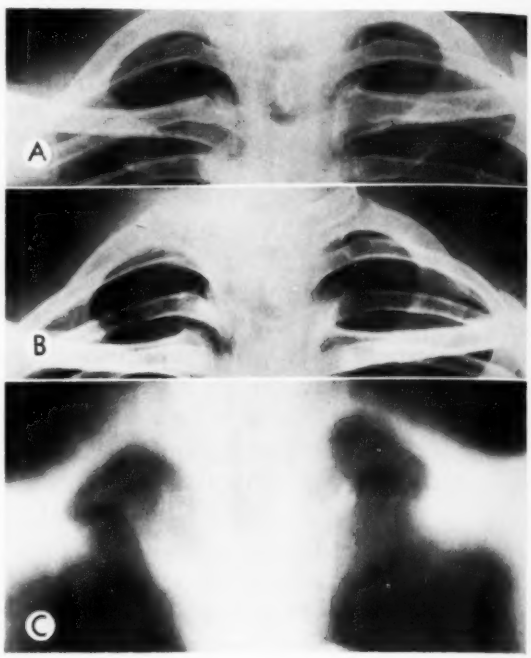

FIG. 1. (*A*) In the right apex, the innominate artery and its bifurcation into the subclavian and common carotid artery are visualized. In the left apex, the first part of the subclavian artery shows calcification in its wall. Because of the angle of projection, the second part is obscured by the second rib. (*B*) In the same patient as *A*, but using a different angle of projection with the tube centered lower, the innominate artery can be recognized in the third intercostal space. Its bifurcation is obscured by the third rib. The right subclavian artery produces a "haziness" in the second intercostal space. The left subclavian artery is well visualized in the second intercostal space. Compare with the left subclavian artery in *A*. (*C*) A laminagram in which both subclavian arteries are visualized also shows origin of the left internal thoracic (mammary) artery as a prominence on the lateral aspect.

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anterior border of the triangular clear space formed with the spine and the aortic arch and seen in this view.

Some of the standard textbooks and monographs[2,6,12,13] of diagnostic roentgenology describe and identify the shadow of the subclavian artery in accordance with Assmann's suggestion, others[7,9–11] seem to ignore it. Caffey's[3] authoritative textbook on pediatric roentgenology describes and illustrates a shadow as "the companion shadow of the left second rib" in the left apex which, by Assmann's criteria and by the observations of the present study, is considered to represent the subclavian artery. Caffey considers this "companion shadow" to be the product of the heavy subcostal muscles on the internal aspect of the left second rib. The "companion shadow," first described by Albers-Schönberg[1] (1908), has been a controversial subject in the literature. According to one concept[4] it represents the proximal border of the pulmonary apex; another explanation[5] is that the shadow is produced by extrapleural structures (subcostal muscles, the last cervical sympathetic ganglion, the supreme thoracic artery and vein and the first thoracic nerve). Apparently the shadow produced by the subclavian artery, as suggested in the present paper, was also interpreted as the "companion shadow of the second rib."

The present paper has two purposes: (1) to re-emphasize the accuracy of Assmann's original observation that the second portion of the subclavian artery may be visualized in the projection of the pulmonary apex in the posteroanterior view of the chest; and (2) to record additional observations.

GROSS AND ROENTGEN ANATOMY

The left subclavian artery is a branch of the aorta and the right is a branch of the innominate artery. The first part of the left subclavian artery runs posteriorly and somewhat laterally to the thoracic portion of the common carotid artery. This segment together with the common carotid artery is commonly seen in the chest roentgenogram just lateral to the translucent air column in the trachea. Then the left subclavian artery turns toward the left. The right subclavian artery reaches a somewhat higher level than its counterpart; nevertheless, this vessel is considerably shorter than the left because of its different origin. The proximally most prominent part of the subclavian artery is located behind the scalenus anterior muscle. From here the artery continues as far as the lateral border of the first rib to become the axillary artery. Thus the scalenus anterior muscle divides the subclavian artery into three parts, the second being the one covered by this muscle. This portion measures approximately 2 cm. in length and is mainly responsible for the shadow in the pulmonary apex. The rest of this shadow is made up of the adjacent portions of the first and third parts of the artery.

Visualization on the roentgenogram is caused by a groove of variable depth in the apex of the lung. This groove decreases the sagittal diameter of this area of the lung compared to that of the adjacent portions of lung and determines the density of the contrasting shadows on the roentgenogram. The difference in the depth of the groove in different persons is one of the reasons why the shadow may be demonstrated in one person and not in another. The less frequent incidence of a visible vascular shadow on the right is the result of the asymmetry of the anatomy of the lungs. The groove on the right side is generally more shallow than that on the left; the relatively higher position of the second part of the artery on this side probably represents a contributing factor. Occasionally, on roentgenograms of excellent quality, particularly on laminagrams, the origin of the internal thoracic artery (internal mammary artery) can be seen on the inferior aspect (Fig. 1*C*). This branch of the subclavian artery arises from the inferior surface of the first part opposite the thyrocervical trunk.

The caliber of the subclavian artery has

been determined by angiocardiographic studies; the average is given as 0.9 cm.[8] Measurements performed in a small number of our cases on conventional chest roentgenograms showed a range of 0.9–1.1 cm., the average being approximately 1.0 cm. This somewhat higher average is explained by the fact that these measurements include not only the lumen but the wall of the vessel as well.

A clear definition of the subclavian artery can be obtained only when both the superior and inferior contours are visualized. In cases where the two contours are obscured by the ribs or are poorly defined because of the angle of projection, the shadow presents itself as a "diffuse haziness" (Fig. 1*B*). The demonstration of the subclavian artery is greatly influenced by the anatomy of the thorax, the width of the intercostal spaces, and the projection angle employed. It was found that a slight cephalad angulation of the central beam (with the tube placed in a lower position) results in better visualization of the subclavian artery in the posteroanterior view than by the usual method (Fig. 5, *A* and *B*). A similar result can be obtained in the anteroposterior view by using a caudally tilted tube, a technique originally employed by Albers-Schönberg for visualization of the pulmonary apex. The same projection is used for anteroposterior roentgenograms of the cervical spine, and the subclavian artery is commonly depicted on these roentgenograms if the pulmonary apex is included.

CORRELATIVE ROENTGEN FINDINGS

Although Assmann's autopsy study and modern angiocardiographic observations locate the subclavian artery where the questionable shadow can be seen on plain roentgenograms, the identification cannot be made with absolute certainty. In the present study we collected material other than angiocardiographic studies with the purpose of adding further support to this concept and possibly eliminating any doubt that the apical shadow in question represents the subclavian artery.

1. Calcium salt deposition in one part of the wall (the media) of the subclavian artery may produce a contrast effect similar to that of an angiocardiographic study (Fig. 2). In the case where the calcium salt deposition is not evenly distributed, the direct continuity of the calcific shadow with the soft tissue shadow of another part of the artery identifies the latter as the subclavian artery (Fig. 3, *A* and *B*). By the dotted line and the plaques produced by the calcification, nature provides an illustration comparable to an anatomic drawing. This "illustration" provides more convincing evidence than any contrast study could produce.

2. In pneumothorax the subclavian artery is visualized against the background of the air-containing pleural cavity (Fig. 4). Because of the increased contrast, the artery is seen more clearly than in a normal chest, although the physical factors are the same since the sagittal diameter of the pneumothorax cavity at the level of the subclavian artery is shorter than that of the adjacent area because of the persistence of the groove on the parietal pleura after the collapse of the lung. In emphysema, pneumatocele and cystic disease a similar situation may exist, the contrast being effected

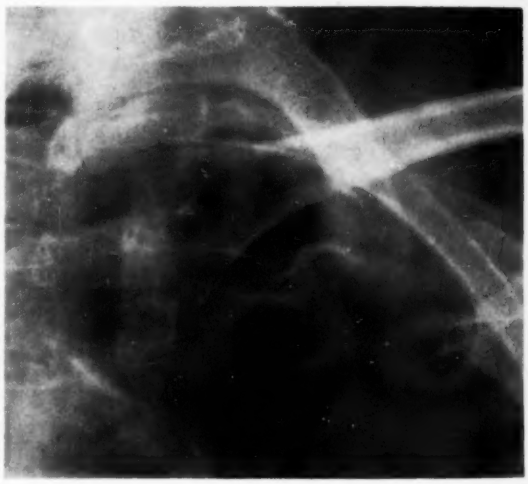

FIG. 2. Extensive calcification in the wall of the left subclavian artery. Note that both the first and the second parts of the artery can be identified.

by the proportion of lung tissue to the air content of the lung in the area of the apex (Fig. 5, *A* and *B*). In any infiltrative process of the pulmonary apex the contrast decreases and the subclavian artery shadow becomes obscured.

3. Laminagraphy is an excellent method for demonstration of the subclavian artery (Fig. 1*C*). The shadow in question is best visualized in cuts where the groove of the subclavian artery is located in the anterior aspect of the pulmonary apex. This cut is usually at a distance of $9\frac{1}{2}$–11 cm. from the posterior wall. Demonstration of the artery is more frequent by this technique than by conventional roentgenography.

CLINICAL SIGNIFICANCE

The shadow of the subclavian artery may be incorrectly interpreted as pulmonary infiltration or thickening of the apical pleura. If calcification is present in the wall of the vessel, this may erroneously be considered to be due to tuberculosis or histoplasmosis. In doubtful cases an effort should be made to clearly define the

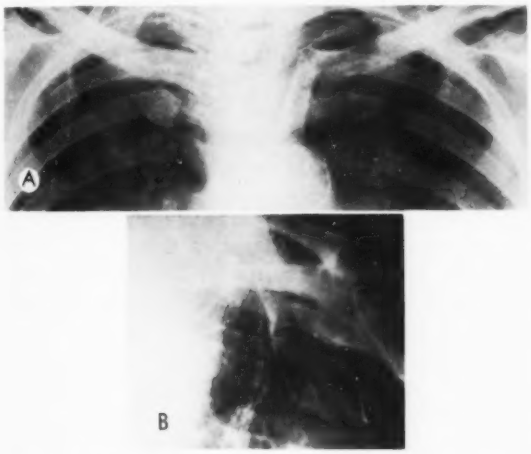

Fig. 3. (*A*) Posteroanterior and (*B*) lateral roentgenograms showing calcification in the wall of the left subclavian artery. In the second part the calcification mainly involves the superior wall, whereas the inferior aspect of the subclavian artery is seen as a "soft tissue shadow." This combination of findings and the lateral view identify the "soft tissue shadow" as that of the subclavian artery. The irregular calcification in the right apex is probably that of the right subclavian artrey.

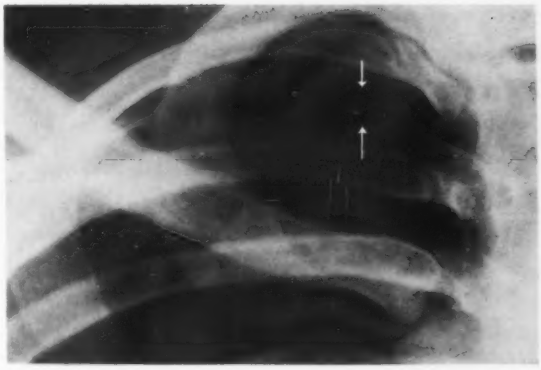

Fig. 4. A right pneumothorax. Note the good delineation of the right subclavian artery.

vascular shadow in an attempt to confirm or to rule out disease of the lung or the pleura (Fig. 6).

On the right side, visualization of the subclavian artery may be helpful in identification of the innominate artery. This may be of importance in the differential diagnosis between an elongated or dilated innominate artery and masses of the superior mediastinum. Thus an unnecessary surgical procedure undertaken on the basis of a

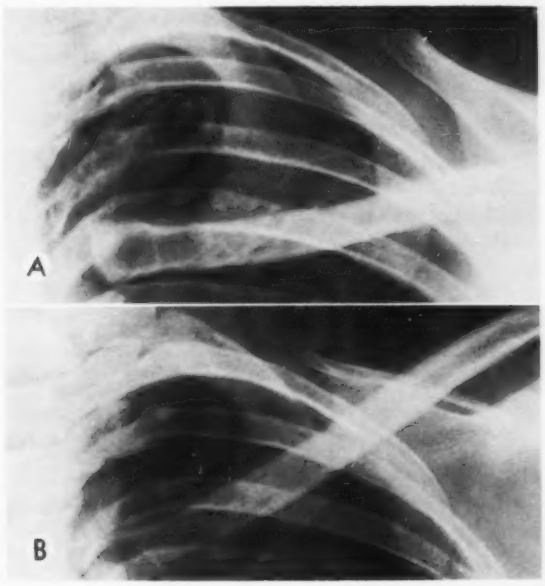

Fig. 5. Cystic disease of the left lung. Note that both the first and second parts of the left subclavian artery are visualized as one continuous structure. (*A*) The artery is clearly visualized in the third intercostal space. (*B*) Using a different projection angle with the tube higher, the artery is partially obscured by the second rib.

FIG. 6. Thickening and a calcification of both apical pleurae. Since the shadows can be followed laterally beyond the medial border of the first rib, this differentiates this finding from shadows of the subclavian arteries.

diagnosis of an "intrathoracic" thyroid can be avoided.

SUMMARY

The subclavian artery (one or both) can be visualized as a curved, band-like soft tissue shadow in the pulmonary apex in about 40 per cent of plain chest roentgenograms. This was first demonstrated by Assmann.[2] Additional observations reported in the present study (laminagrams, plain roentgenograms of cases of calcification in the wall of the artery, pneumothorax, pneumatocele, etc.) provide further support for this concept. Incorrect interpretation of the shadow in question may lead to erroneous diagnosis of apical infiltration or pleural or parenchymal calcification.

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The author wishes to express his appreciation to Dr. Marion Faris for her cooperation in the statistical study of this paper.

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Vol. 86, No. 6

CINEFLUOROGRAPHY OF CORONARY ARTERY CALCIFICATION*

CORRELATION WITH CLINICAL ARTERIOSCLEROTIC HEART DISEASE AND AUTOPSY FINDINGS

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CINEFLUOROGRAPHY of the heart is the best roentgenologic method for detecting coronary calcification,[8] usually considered direct evidence of coronary artery disease. The purpose of this study was to find any correlation between clinically manifested arteriosclerotic (ischemic) heart disease and coronary calcification as seen at cinefluorography of the heart. Previous roentgenologic studies[7,9,13,16,17,20] without the aid of cinefluorography have been unsuccessful in this respect.

HISTORY

Lenk[9] in 1927 first demonstrated a calcified coronary artery on a chest roentgenogram of a living patient, a sixty-eight year old man with a left ventricular aneurysm and no cardiac symptoms. He hoped that with the Bucky technique and short exposures more coronary calcifications would be seen.

Wosika and Sosman[20] in 1934 reported 2 men and 1 woman with roentgen evidence of calcified coronary arteries, all of whom had angina.

Snellen and Nauta[16] in 1937 collected 40 cases of coronary calcifications; there were 32 men and 8 women, ranging in age between fifty-seven and eighty-five years. Three had calcification of both right and left coronary arteries; 37 had calcification of the left coronary artery only. They used no special techniques or positions, but relied on routine posteroanterior and oblique views of the chest. Seventeen of their patients had cardiac symptoms, mostly angina. The authors felt they could not judge the clinical value of detecting coronary calcification by roentgenography because of the way they had selected their patients.

Sosman[17] in 1943 discussed the technique for locating and identifying pericardial and intracardiac calcifications. He was of the opinion that roentgen evidence of coronary sclerosis did not imply angina but indicated a specific type of damage in a specific place and could be reported only as such. Over a ten year period he found 42 coronary calcifications by fluoroscopy.

Habbe and Wright[7] in 1950 used multiple, high-speed spot roentgenograms to detect coronary calcifications. Over a two and one-half year period they examined every cardiac patient over the age of forty years, taking a minimum of four spot roentgenograms in different positions, and found 28 such cases, 3 per cent of the total cases examined by this method. They added kymography when necessary. They believed that by their method they found twice as many cases as is possible by fluoroscopy alone.

Reich and Witten[13] in 1957 reported 6 patients (5 men and 1 woman) with left coronary artery calcifications. They found the most common site of calcification to be beneath the left heart border in the region of the upper left ventricular contour. Two of their patients had angina and 1 had a coronary occlusion, although 5 of the 6 had negative electrocardiograms.

MATERIALS AND METHODS

Our material consisted of two groups of predominantly white male Veterans Ad-

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Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

ministration Hospital patients, a control group of patients without detectable heart disease and a larger "heart disease group" with suspected or evident cardiac disease including ischemic heart disease.

The control group was made up of 60 men, 15 in each decade from ages forty to eighty, with normal hearts as far as could be determined by histories, physical examinations, electrocardiograms, and chest roentgenograms. These patients were hospitalized for various noncardiac causes. No one with hypertension or diabetes mellitus was included. All underwent cinefluorography for the purpose of detecting coronary calcification.

The heart disease group consisted of 630 consecutive patients, predominantly male, referred to the radiology department for cinefluorography of the heart for suspected or known heart disease of various types. Practically all were cases of acquired heart disease. Cinefluorograms were obtained during the nineteen month period between January 1, 1958 and July 31, 1959, and those showing coronary calcification on the initial reading were reviewed and two factors were studied:

(1) The distribution of calcification between the three main coronary branches, the anterior descending and circumflex branches of the left coronary artery and the right main coronary artery, was ascertained. Due to the difficulty in deciding whether a collection of calcium was in the main stem of the left coronary artery or in the anterior descending branch, all of this calcium was considered to be in the anterior descending branch, since it has been shown that the most common point of calcification is in that branch just distal to the bifurcation of the main left coronary artery.[1,12,15] Calcium in the circumflex branch was usually easy to identify and to locate accurately.

(2) The degree of calcification was determined. This was graded by 2 observers on a one to four plus scale.

The clinical records of all of the heart disease group patients in whom coronary calcification was revealed by cinefluorography were then reviewed for the presence of angina, electrocardiographic evidence of old or new myocardial infarction, and a clinical diagnosis of arteriosclerotic heart disease. The latter diagnosis was reached by the ward physician in practically all cases by considering the usual clinical criteria for that particular type of heart disease and independent of the finding of coronary calcification at cardiac cinefluorography. The same evaluation was made of the records of 80 patients without coronary calcification chosen at random from the heart disease group and the clinical findings of both subgroups were compared. There were 5 women in these two subgroups.

Twenty-one autopsy protocols of patients who had had cardiac cinefluorographies were reviewed. The presence or absence of coronary calcium on each cinefluorogram was correlated with the presence or absence of coronary atherosclerosis and myocardial fibrosis at postmortem examination in the same patient.

The cinefluorographic unit consisted of a Westinghouse five inch image intensifier equipped with a 16 mm. Kodak motion picture camera. The film was Kodak Linagraph Shellburst film in one hundred foot rolls developed in standard x-ray solutions and dried in air. The cinefluorograms were viewed with a 16 mm. Bell and Howell projector.

Our technique of cardiac cinefluorography,[8] briefly described, consists of six film sequences: (1) in the posteroanterior view, the field sweeps along the right heart border from the diaphragm to the superior vena cava and (2) along the left heart border from the aortic arch down to the left hemidiaphragm; (3) in the right anterior oblique projection, we record from the diaphragm to the pulmonary arteries and then down the left heart border to the diaphragm again; (4) in the left anterior oblique position, the right atrium, the ascending aorta and the mid-cardiac mass are scanned, followed by (5) a record of the posterior border of the heart; finally (6) in the right lateral view a recording of the

posterior border of the heart and the mid-cardiac mass is again obtained.

Usually 30 to 40 feet of film, at the rate of 15 frames per second will record a routine cardiac cinefluorographic examination. At 110 kv., the ma. will fluctuate between 5 and 15 for an average-sized individual. Total radiation exposure averages about 20 r in air, delivered to a constantly changing skin field through a portal 4.75 inches in diameter.

RESULTS

Table I compares by decades the incidence of coronary calcification between the control and heart disease groups. Both groups showed a steady rise in coronary calcification with age up to a peak in the sixty to sixty-nine year group, followed by a drop in the seventy to seventy-nine year group. The slight differences in the percentages between the control and heart disease groups are not significant. Both groups showed an average incidence of coronary calcification of 25 per cent in the forty to seventy-nine year age range. Of the 630 known or suspected heart disease patients on whom cinefluorographies were performed, 150 or 23.8 per cent showed coronary calcification.

Table II shows the age distribution of

TABLE I

COMPARISON OF INCIDENCE OF CORONARY CALCIFICATION BETWEEN CONTROL AND HEART DISEASE GROUPS BY DECADES

Age Groups	Control Group* 60 Patients (per cent)	Heart Disease Group† 630 Patients (per cent)
20–29		0
30–39		9
40–49	13	8
50–59	27	22
60–69	40	36
70–79	20	32
80–89		40

* No detectable heart disease.

† Suspected or known heart disease of various types including arteriosclerotic.

TABLE II

AGE DISTRIBUTION OF PATIENTS IN HEART DISEASE GROUP USED FOR CLINICAL CORRELATION STUDY

Age Groups	Coronary Calcification: No. in Each Age Group	Coronary Calcification: Per Cent in Each Age Group	No Coronary Calcification: No. in Each Age Group	No Coronary Calcification: Per Cent in Each Age Group
20–29	0	0	5	6
30–39	7	5	12	15
40–49	8	5	15	19
50–59	22	15	13	16
60–69	92	61	28	35
70–79	17	11	6	8
80–89	4	3	1	1
	150*	100	80†	100

* Two females were included in this group.

† Selected at random from those patients in the heart disease group in whom no coronary calcification was demonstrated. Three females were included in this group.

the 150 patients with coronary calcification and the 80 randomly selected patients without calcification, all within the heart disease group. The sixty to sixty-nine year age bracket contained the largest number of patients in both groups. There were more younger patients among those without coronary calcification than among those with calcium. This produced an average age difference of about ten years (62.4 years as compared to 52.5 years) between the two groups. A comparison of the signs and symptoms of ischemic heart disease between these groups was, therefore, made by decades rather than by total numbers.

Table III compares those patients with coronary calcification and those in whom no calcification was found with respect to incidence of angina, electrocardiographic evidence of old or new myocardial infarction, and clinical diagnosis of ischemic heart disease. Age groups twenty to twenty-nine and eighty to eighty-nine were omitted because of an insufficient number of patients in those decades. The table shows a substantially higher incidence or arteriosclerotic heart disease in all age groups in those

TABLE III

INCIDENCE OF ANGINA, ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL INFARCTION AND CLINICAL DIAGNOSIS OF ARTERIOSCLEROTIC HEART DISEASE IN PATIENTS WITH AND WITHOUT CORONARY CALCIFICATION AT CINEFLUOROGRAPHY

Age Groups*	Calcification Seen (150 Patients)			No Calcification Seen (80 Patients)		
	Angina (per cent)	Infarct† (per cent)	Arteriosclerotic Heart Disease (per cent)	Angina (per cent)	Infarct‡ (per cent)	Arteriosclerotic Heart Disease (per cent)
30–39	42.9	28.6	57.1	8.3	8.3	8.3
40–49	50.0	50.0	50.0	13.3§	0.0	0.0
50–59	36.7	18.9	45.5	30.8	15.4	38.5
60–69	41.3	31.5	60.9	21.4	17.8	32.1
70–79	41.2	35.3	52.9	0.0	0.0	16.7

* Age groups 20–29 and 80–89 omitted because of insufficient number of patients in these decades.
† No electrocardiogram available in 4 patients.
‡ No electrocardiogram available in 3 patients.
§ Represents 2 patients with angina due to rheumatic aortic stenosis.

cases where coronary calcium was demonstrated at cinefluorography. This difference was particularly high in the younger age brackets, thirty to thirty-nine and forty to forty-nine years.

Table IV shows that the incidence of clinically manifested ischemic heart disease rises in direct relation to the number of coronary branches exhibiting calcium on cinefluorograms. The amount of coronary calcium in any one branch appeared to show no definite correlation with the presence of clinical arteriosclerotic heart disease.

Table V shows the distribution of calcium among the three main coronary branches in 150 patients with coronary calcifications. The left anterior descending branch was the most frequent site of coronary calcification; the right main artery and the left circumflex branch were second and third, respectively. Most of the calcium was usually confined to the proximal por-

TABLE V

DISTRIBUTION OF CALCIUM AMONG CORONARY ARTERIES AS SEEN AT CINEFLUOROGRAPHY IN 150 PATIENTS

Artery	No. of Times Calcified
Left anterior descending artery	136*
Right main coronary artery	59
Left circumflex artery	25
Two arteries	36
Three arteries	13

* Includes calcium in left main coronary artery.

TABLE IV

RELATION OF EXTENT OF CORONARY CALCIFICATION AT CINEFLUOROGRAPHY TO INCIDENCE OF CLINICAL DIAGNOSIS OF ISCHEMIC HEART DISEASE

No. of Coronary Arteries Calcified at Cinefluorography	No. of Patients	No. of Patients with Arteriosclerotic Heart Disease	Per Cent
One	101	53	52
Two	36	23	64
Three	13	11	83
Total	150		

tion of each artery. Two main branches were calcified in 36 patients and all three in 13 patients.

Table VI is a correlation of the presence of calcification at cinefluorography with the degree of coronary atherosclerosis found at autopsy in 15 patients. Table VII shows the correlation between the absence of calcification at cinefluorography and the state of the coronary arteries and the myocardium at necropsy in 6 patients. Table VI shows the exceptionally high correlation of coronary calcium seen *in vivo* with moderate to severe coronary atherosclerosis at postmortem examination. Table VII, on the other hand, indicates that the absence of coronary calcium appears to point to normal coronary arteries. The tables also show that heart enlargement did not appear to interfere with the cinefluorographic recognition of coronary calcium as much as expected.

DISCUSSION

In order to demonstrate even slight amounts of coronary calcification and as many calcifications as possible, a definite sequence of cinefluorography, with all the positions outlined previously, should be used. It is important in ascertaining the presence of coronary calcification to scrutinize the location, movement and form of the calcific deposits.[4,5,8,16]

Examples of location and movement are shown in Figures 1–4. The form of calcium deposition varies from long densities composed of parallel interrupted lines resembling sclerotic extremity arteries to the fleck of calcium seen in a minimally calcified left coronary artery when it is foreshortened in the left anterior oblique view. The curvilinear character of coronary calcifications aids in their detection.

Although with cinefluorography the detection of coronary calcification has been greatly improved as shown by our discovery of coronary calcium in 25 per cent of a group of males without heart disease, the method by no means visualizes all coronary calcium present. Blankenhorn and Stern[3] found that 80 per cent of 76 patients, the majority of whom were over forty years of age and had died of various cardiac and noncardiac causes, showed coronary calcium on roentgenograms of the heart after removal from the cadaver.

The calcium which is demonstrated by cinefluorography seems to consist of larger plaques, probably at least 2 to 3 mm. in size,[3,19] and, judging from our autopsy series as well as other studies,[15,18] its presence indicates considerable intimal atherosclerosis and coronary stenosis at the points of calcification.

There are many factors aside from the small size of the calcification which may be responsible for nondetection of coronary calcification by cinefluorography.[8] These are obesity, cardiomegaly, pulmonary congestion and effusion, pulmonary and pleural disease of noncardiac origin, failure to examine the heart in all positions, and inattentiveness or inexperience on the part of the observer.

The increasing incidence of coronary calcification with age is well known[10,11] and is shown in Table I. The drop in incidence between the seventh and eighth decades in our series, if real, makes one speculate that an increased death rate from ischemic heart disease during the seventh decade may have produced this drop and that perhaps this decade represents a critical period in those with coronary calcification.

We found a much higher prevalence of clinical ischemic heart disease in patients with coronary calcification than in those without calcium (Table III). This difference was even higher than expected. Fifty-eight per cent of 150 patients showing coronary calcium on cinefluorograms were diagnosed as having clinical ischemic heart disease as compared with 39 per cent of a comparable group without coronary calcification.

The direct relationship between the number of calcified coronary branches found at cinefluorography and the incidence of arteriosclerotic heart disease (Table IV) was expected. Not only did 11 of 13 patients

TABLE VI

CORRELATION OF PRESENCE OF CORONARY CALCIFICATION AT CINEFLUOROGRAPHY WITH STATE OF CORONARY ARTERIES AND MYOCARDIUM AT AUTOPSY

No.	At Cinefluorography				At Autospy		
	Age	Heart Size	Degree of Calcification		Left Coronary Artery	Right Coronary Artery	Myocardium
			Left	Right			
1	66	Generalized enlargement	0	2+	80% occlusion of anterior descending artery; complete occlusion of circumflex	Fresh occlusion 6 cm. from ostium	Scarring
2	64	Left ventricular enlargement	0	2+	Marked atherosclerosis of coronary arteries with numerous areas of pin-point narrowing		Fresh infarct in septum; fibrosis of left ventricular myocardium
3	66	Moderate enlargement	1+	0	Marked atherosclerosis of anterior descending and circumflex arteries	Relatively uninvolved	Complete loss of myocardium along lateral wall of left ventricle
4	68	Generalized enlargement	1+	0	Anterior descending and circumflex arteries densely calcified; maximum narrowing in anterior descending artery 40% of original lumen	Moderate sclerosis; narrowed to 15% of normal	Diffuse fibrosis of myocardium
5	69	No enlargement	1+	2+	Minimal sclerosis at bifurcation; otherwise normal	Ostium 50% narrowed by sclerosis	Normal
6	65	Left ventricular aneurysm	2+	0	Coronary arteries mildly sclerotic but wide open		Normal
7	64	Severe enlargement	1+	0	Moderate to severe atherosclerosis in all branches with scattered calcium in all branches		Thinning over distal two-thirds of lateral wall of left ventricle
8	65	No enlargement	1+	0	80% obliteration of anterior descending artery 2 cm. from ostium; circumflex 50% occluded	80% occluded 2 cm. from ostium	Flabby; microscopic study showed focal scarring and loss of cardiac muscle
9	64	Moderate left ventricular enlargement	1+	0	Lumen of anterior descending artery about 10% of normal; circumflex least affected	Narrowed by 50%	Extensive fibrosis in lower two-thirds of septum and apex
10	60	Left ventricular enlargement	2+	1+	Calcification in anterior descending and circumflex arteries about 4 cm. in length	Calcification in media for 4 cm. beyond ostium	Thinning at apex of left ventricle about 4 cm. in diameter
11	62	Generalized left ventricular enlargement	1+	0	Anterior descending artery severely sclerotic	Normal	Apex of left ventricle thinned to 4 mm.
12	69	Slight enlargement	3+	1+	Coronary arteries showed considerable amount of calcification in their walls but the lumina not appreciably narrowed		Three small patches of hemorrhagic softening in left ventricular wall
13	65	No enlargement	2+	0	Minimal calcification in first 2 cm. of anterior descending branch	Atherosclerosis but no calcium	Negative
14	62	Left ventricular enlargement	1+	1+	Severe calcification of both coronary arteries with marked narrowing but no occlusion		Slight softening of left ventricle posteriorly
15	44	Left ventricular enlargement	1+	0	Coronary arteries widely patent throughout; calcification and stenosis of aortic valve		Normal

TABLE VII

CORRELATION OF ABSENCE OF CORONARY CALCIFICATION AT CINEFLUOROGRAPHY WITH STATE OF CORONARY ARTERIES AND MYOCARDIUM AT AUTOPSY

No.	At Cinefluorography		At Autopsy		
	Age	Size of Heart	Left Coronary Artery	Right Coronary Artery	Myocardium
1	59	Generalized enlargement	Anterior descending artery markedly sclerotic; lumen of circumflex less than 50% of normal	Extreme atherosclerosis	Large infarct in posterior wall of left ventricle
2	40	No enlargement	Normal	Normal	Normal
3	71	Generalized enlargement	Coronary arteries very patent with no evidence of atheromatous degeneration		Normal
4	51	No enlargement	Normal	Normal	Normal
5	31	Generalized enlargement	Normal	Normal	Thinning of interventricular septum
6	65	Enlarged	Coronary arteries patent except for small segmental narrowing of anterior descending branch about 1.0–0.5 cm. from ostium		Normal

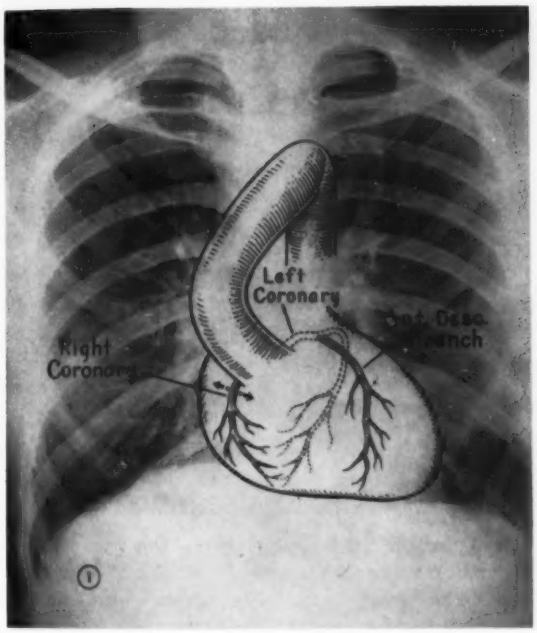

FIG. 1. Posteroanterior view showing the movement and position of the coronary arteries.

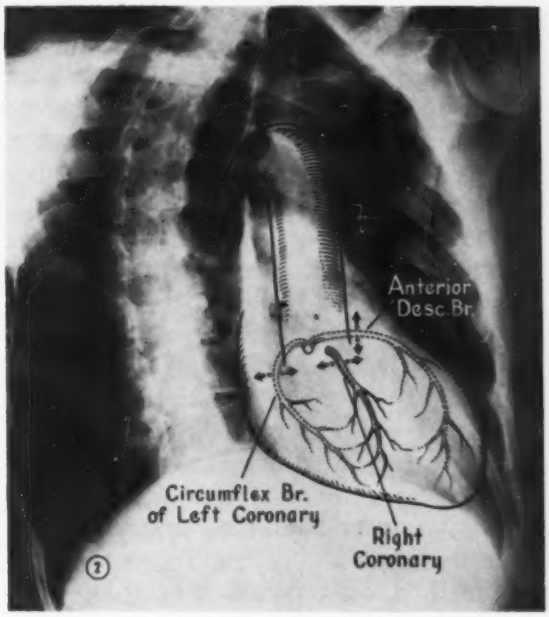

FIG. 2. Right anterior oblique view showing the position and movement of the coronary arteries.

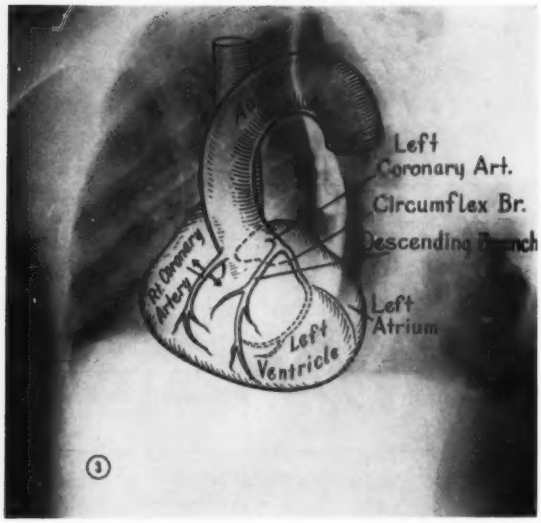

FIG. 3. Left anterior oblique view showing the position and movement of the coronary arteries.

(83 per cent) with all three major branches calcified carry a clinical diagnosis of arteriosclerotic heart disease (in contrast to an over-all arteriosclerotic heart disease incidence of 58 per cent in the 150 patients with coronary calcium), but these 13 patients also had an infarct incidence of 50 per cent (in contrast to an over-all infarct percentage of 30.7 in the 150 patient group). This observation could represent an important factor in the individual prognostication of the risk of arteriosclerotic heart disease and myocardial infarction when using cinefluorography as a screening method.

The left anterior descending coronary artery was found to be the most commonly calcified branch (Table v), which agrees with autopsy studies.[3,12,15]

Autopsy studies shown in Table vi indicate that 13 of 15 patients with coronary calcium seen at cinefluorography had moderate to severe atherosclerosis of the coronary arteries. Of the remaining 2 patients, one had mild coronary sclerosis while the other had normal coronary arteries but had calcification and stenosis of the aortic valve. This last case probably represents either an error in locating the calcium on the part of the radiologists, or a negative error on the part of the pathologist.

Most of the patients in whom moderate to severe coronary atheromatosis was found at autopsy also had marked narrowing of one or more coronary branches. Ten of the 13 patients had localized or diffuse myocardial fibrosis. Ten also had cardiac enlargement of various degrees which apparently did not interfere with the detection of coronary calcium at cinefluorography.

A recent pathologic study of 667 postmortem hearts of individuals, fifteen years of age and older at a New York State hospital, also revealed a relationship between the presence of coronary artery calcification and myocardial infarction, as well as increasing probability of infarction with an increase in the number of coronary arteries calcified.[14]

To further attest to the accuracy of cine-

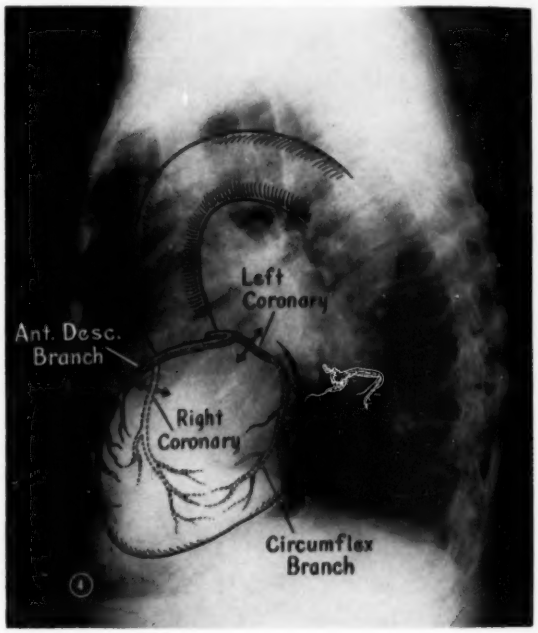

FIG. 4. Left lateral view showing the position and movement of the coronary arteries. The right coronary artery is usually anterior to the anterior descending branch of the left coronary artery in the left lateral view. In the case illustrated here an abnormally large left ventricle displaces the left coronary branch anteriorly.

fluorography in detecting coronary artery disease, 5 of 6 patients (Table VII) with known or suspected heart disease but with no calcium revealed at cinefluorography were found to have normal coronary arteries and myocardium at autopsy. In only one patient was severe coronary atherosclerosis observed at necropsy which could not be detected during cinefluorography of the heart *in vivo* even after review of the cinefluorogram. However, generalized cardiomegaly was present in this patient.

We plan to follow those patients in this study in whom coronary calcification was revealed at cinefluorography but who, as yet, do not exhibit the signs and symptoms of ischemic heart disease. We will then compare them with a similar group of patients without demonstrable coronary calcification with respect to time and manner of onset of clinical arteriosclerotic heart disease.

Since coronary arteriography at present is still unsuitable for routine use due to the complexity of the equipment and the hazard to the patient,[2,6] the relatively simple, rapid, and innocuous method of cinefluorography may be the most efficient screening procedure for the detection of potential candidates for arteriosclerotic heart disease at a time when no electrocardiographic changes or signs and symptoms are present to indicate the extent of coronary atherosclerosis.

SUMMARY AND CONCLUSIONS

Carefully carried out cinefluorography of the heart revealed a high number of coronary artery calcifications. Cardiac enlargement did not interfere as much as expected.

Autopsies showed that visualization of coronary calcification on cinefluorograms indicated considerable coronary atherosclerosis.

In one-fourth of a control group of 60 male patients between forty and seventy-nine years without detectable heart disease and one-fourth of a group of patients in the same age bracket with known or suspected heart disease of various types including ischemic heart disease, coronary calcification was revealed at cinefluorography.

Fifty-eight per cent of 150 predominantly male patients with coronary calcium found at cinefluorography had arteriosclerotic heart disease by clinical evaluation as compared with 39 per cent of a comparable group without coronary calcification.

There was a much higher incidence of angina and myocardial infarction in cases where coronary calcium was detected at cinefluorography.

There was a direct relation between the number of major coronary branches calcified as revealed by cinefluorography and the incidence of clinically manifested arteriosclerotic heart disease. Eleven of 13 men with three major branches calcified had signs or symptoms of ischemic heart disease.

There appears to be a high correlation between coronary calcification as detected by cinefluorography and clinically manifested arteriosclerotic heart disease in males if heart disease is evident or suspected.

Although cinefluorographic evidence of coronary calcium *by itself* appears to be unreliable for predicting the presence of symptomatic ischemic heart disease, it may prove to be a good indicator of potential ischemic myocardial disease.

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IDIOPATHIC MYOCARDIAL HYPERTROPHY

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IDIOPATHIC myocardial hypertrophy refers to cardiac enlargement of unknown etiology in relatively young adults. Usually, a severe grade of congestive failure is present; however, some cases may present with only minimal signs of failure, while in others the course is dominated by the effects of pulmonary and systemic embolization. By definition, the diagnosis ultimately rests on the exclusion of known cardiac disorders, although it has been shown that a presumptive diagnosis may be suggested from the clinical picture.[8] It is the purpose of this paper to call attention to certain previously undescribed roentgenographic features which are of great aid in the differential diagnosis of this disease.

MATERIAL AND METHODS

An analysis of 21 cases of idiopathic myocardial hypertrophy yielded 13 cases with satisfactory chest roentgenograms.[8] Four new cases were added to the group. Necropsy confirmation was obtained in 7 cases.

Most of the chest roentgenograms available were posteroanterior teleroentgenograms. These were analyzed for cardiac size, specific chamber enlargement, aortic knob size, status of the pulmonary vasculature, and presence or absence of pulmonary infarction. These findings were then compared with the changes seen in patients with known heart disease of various causes.

CLINICAL FINDINGS

The patients ranged in age from twenty-eight to fifty-seven years with a mean age of forty years. Thirteen were males and 4 were females. Nearly all were Negro, but this is true of the general patient population at our institution.

The symptoms and signs of this group of patients are summarized in Table 1. The most common symptom, dyspnea, is related to congestive heart failure. It is not unusual to obtain a history of cardiac enlargement discovered on routine examination several months to years prior to the onset of symptoms. Although mild antecedent upper respiratory infections are frequently recalled, these patients do not usually have fever or active infection when cardiac manifestations develop. Attempts to identify viral agents by serologic or culture methods have been unsuccessful. Alcoholism may be associated with this disease, but the nutritional status is good and avitaminoses are absent.[4] About 30 per cent of the patients die within one year of the onset of symptoms from intractable congestive heart failure or systemic and pulmonary emboli.

Physical examination may demonstrate

TABLE I

CLINICAL FINDINGS IN 17 PATIENTS

Symptoms and Signs	No.	Per Cent
Dyspnea	16	94
Rales	16	94
Hepatomegaly	13	76
Increased Jugular Venous Distention	13	76
Peripheral Edema	13	76
Chest Pain	8	47
Pleural Effusion	8	47
Pulmonary Embolism	8	47
Hemoptysis	7	41
Alcoholism	5	29
Antecedent Upper Respiratory Infections	5	29
Systemic Emboli	5	29
Ascites	4	24
Digitalis Toxicity	4	24
Myocardial Infarction	1	6

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the signs of congestive heart failure, *e.g.*, rales, hepatomegaly, increased jugular venous distention and peripheral edema, or may be completely negative except for cardiac enlargement. The point of maximum impulse is usually diffuse and forceful; it is located outside the midclavicular line. The rhythm is generally a sinus tachycardia, but slows with digitalis therapy. Gallop rhythm is found in about 50 per cent of cases; organic murmurs are not heard.

The electrocardiogram characteristically shows sinus rhythm and signs of left atrial and left ventricular enlargement. Cardiac catheterizations, ballistocardiograms, and pulmonary function tests are consistent with congestive heart failure.[8]

PATHOLOGIC FINDINGS

All 7 autopsied cases showed pancardiac hypertrophy and dilatation. The weights of the hearts ranged from 500 to 960 gm. with a mean of 600 gm. The pericardium was normal in all instances. The coronary arteries were widely patent and no occlusions could be identified. Only one case showed moderate atherosclerosis of the right coronary artery. The cardiac valves were normal except for functional dilatation of the rings. The endocardium was thin and smooth except in 4 cases where it underlay mural thrombi. These thrombi were located in the left ventricle in all cases, and, in addition, in the right ventricle in one case. The aortas were normal or showed minimal patchy atherosclerosis. Neither renal nor systemic vascular abnormalities suggested pre-existing hypertension. Emboli or infarction were distributed as follows: pulmonary, 4 cases; renal, 3 cases; splenic, 2 cases; and mesenteric, 1 case.

Histologic sections of the hearts showed myocardial hypertrophy with nonspecific tissue changes. Interstitial fibrosis was focal in 6 cases and primarily subendocardial in one case. Viral cultures of the heart were negative in the 2 cases in which they were performed.

A full discussion of the pathologic anatomy of idiopathic myocardial hypertrophy has been given by Elster and his associates.[4]

ROENTGENOGRAPHIC FINDINGS

A similar roentgenographic picture was found in over three-quarters of our cases. The findings are summarized in Table II. Left ventricular enlargement predominates. The diastolic overload on the left ventricle results in dilatation and elongation of its outflow tract. Thus, there is displacement of the cardiac apex to the left and downwards.

The right ventricle is moderately dilated. This results in prominence of that portion of the left heart border just above the left ventricle. The relatively normal pulmonary artery segment and the counterclockwise rotation of the heart, caused by the markedly enlarged left ventricle, combine to preserve the "cardiac waist" on the posteroanterior roentgenogram. In the presence of pulmonary infarction with acute cor pulmonale, there is a further increase in the size of the right ventricle and pulmonary artery, and the cardiac waist is obliterated (see Fig. 1, *A* and *B*).

TABLE II

ROENTGENOGRAPHIC FINDINGS IN 17 PATIENTS

	No.	Per Cent	
Cardiomegaly	17	100	
Aorta			
Hypoplastic	11	65	77%
Normal	2	12	
Enlarged	4	23	
Left Ventricular Hypertrophy	17	100	
Left Atrial Size			
Normal	9	53	
Minimal increase	6	35	
Moderate increase	2	12	
Right Ventricular Enlargement	16	94	
Right Atrial Enlargement	2	12	
Main Pulmonary Artery			
Normal	11	65	
Increased size	6	35	
Pulmonary Vasculature Increase	6	35	
Pulmonary Infarction	4	23	
Mesenteric Infarction	1	6	

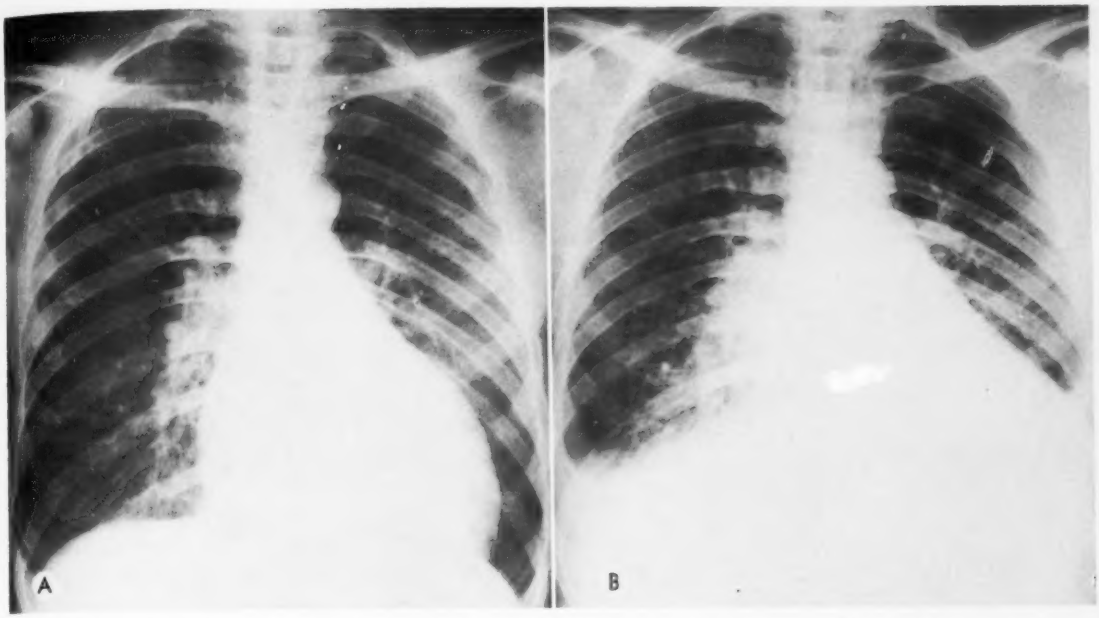

Fig. 1. (*A*) The typical picture of idiopathic myocardial hypertrophy is seen. There is biventricular enlargement and a small aortic knob. Note the preservation of the cardiac waist. (*B*) Same patient as *A*. Pulmonary infarction and acute cor pulmonale with considerable prominence of the pulmonary artery segment and increased right ventricular enlargement.

The left atrium is slightly dilated. Though this was not appreciated on all posteroanterior roentgenograms, the left atrium was seen to be enlarged in all cases of which right anterior oblique roentgenograms with barium in the esophagus were obtained, and in all autopsied cases. The right atrium is also slightly dilated. The cardiac borders are sharply outlined (stenciled heart[2]) due to decreased pulsations of the weakened heart muscle.

The aorta is of normal caliber and the aortic knob appears small relative to the enlarged heart. There is usually no aortic or valvular calcification.* The larger pulmonary arteries are normal; in one-third of the cases they were slightly prominent and were described as top normal. There is no roentgenographic evidence of pulmonary arterial or venous hypertension. The pulmonary veins are normal unless congestive failure or pulmonary infarction supervenes. Pulmonary infarcts were seen in 8 cases.

* Two of our cases showed calcification in the aortic knob. We believe this to be an incidental finding.

Angiocardiograms were performed on 3 patients. There was marked dilatation of the heart, especially of the ventricles. There was pooling of contrast medium on both sides of the heart. This is similar to the pooling and delayed emptying seen in congestive failure. No organic abnormalities were demonstrated.

The following 2 cases were selected to illustrate the characteristic findings of this disease.

REPORT OF CASES

Case 1. This thirty-nine year old Negro female was admitted to Philadelphia General Hospital in March, 1958 with a fifteen month history of gradually progressing congestive failure, not refractory to therapy. Seven weeks prior to admission she had a transient episode of right hemiparesis and loss of speech lasting two hours.

Physical examination on admission revealed a well nourished female in moderate respiratory distress. The blood pressure was 110/88, respirations 24, pulse 100 and temperature 100.4° F. The neck veins were markedly distended even

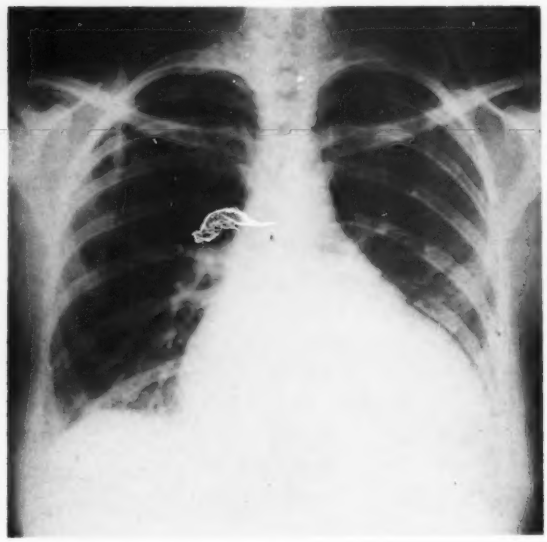

FIG. 2. Case I. There is biventricular enlargement, fullness of the pulmonary artery segment, and a small aortic knob.

at 90°; a hepatojugular reflux was present. The lungs were clear to percussion and auscultation. The apex beat was at the fifth interspace in the left midaxillary line. There was a grade 3, blowing systolic murmur heard to the left of the sternum in the fourth and fifth interspace which became accentuated with inspiration. There was questionable shifting dullness in the abdomen. A smooth liver edge was palpable 5 cm. below the costal margin. There was 2 plus to 3 plus pitting edema of the legs. No clubbing was noted. Neurologic examination was within normal limits.

Extensive laboratory examinations including urinalysis, complete blood cell count, urea nitrogen, serum proteins, serology, antistreptolysin titer, antihyaluronidase titer, gastric washings for acid fast bacilli, sedimentation rate, gum biopsy, L.E. preparations were essentially negative. All serologic tests for viruses were negative except for the Coxsackie neutralization test which showed titers of 1:8, 1:16 and 1:64 on successive weeks. Electrocardiograms showed atrial disease and biventricular hypertrophy. Roentgenograms of the chest showed a large globular heart (Fig. 2) and the typical findings of a small aortic knob with normal pulmonary vasculature. The venous pressure was 165 mm. H_2O; the circulation time (decholin), 47 seconds. Cardiac catheterization was performed and the findings were consistent with congestive heart failure which persisted despite vigorous therapy and she died on the twenty-sixth hospital day.

The clinical diagnosis was idiopathic myocardial hypertrophy, Coxsackie myocarditis or rheumatic carditis.

Necropsy. The pleural, pericardial and peritoneal cavities were free of abnormal collections of fluid. The heart weighed 500 gm. and was globular in shape. The myocardium was firm, brownish and homogeneous throughout. The papillary muscles were flattened. The endocardium was smooth and transparent, except for an area underlying a mural thrombus in the anterior wall of the left ventricle. This thrombus measured 3×5 cm. and extended from the heart to near the lower margin of the aortic valve. All four cardiac chambers, most notably the left ventricle, were markedly dilated. The valves were thin, delicate and normal in number. The tricuspid orifice admitted three and one-half fingers. The coronary ostia were patent; the coronary vessels were normally distributed, thin-walled and patent throughout. The aorta and its main branches were lined by a smooth intima. The right lung weighed 350 gm., the left 280 gm. The liver weighed 1,410 gm. and was congested. The kidneys showed only old infarcts. No other significant abnormalities were present.

Histologic examination of the heart muscle showed myocardial hypertrophy and focal interstitial fibrosis. The coronary arteries were normal. The lungs showed passive congestion and pulmonary atherosclerosis. Except for several old infarcts, histologic sections of the kidneys were normal. The other organs were unremarkable.

Viral cultures of the heart were negative.

Comment. The rising titer in the Coxsackie neutralization test suggested current virus infection. However, the patient's symptoms and cardiac enlargement started long before these tests were performed. Apparently, the Coxsackie infection was merely an incidental finding in this case. This was borne out by the negative cultures for viruses from the heart muscle and the absence of inflammatory changes.

CASE II. This thirty year old husky Negro laborer was admitted to Philadelphia General Hospital for the first time in September, 1958 because of extreme dyspnea. He stated that he

was in good health until July, 1958 when he noted gradual progressive dyspnea which was incapacitating by September, 1958. He denied prior sore throat, rheumatic fever, hypertension or fever.

Physical examination revealed a well nourished male in acute and chronic respiratory distress. The blood pressure was 110/80, the pulse 120, respirations 32 and temperature 98.6° F. The fundi were normal. The lungs were clear to percussion and auscultation. The left border of the heart was percussed 12 cm. from the midsternal line. The rhythm was rapid and regular. No murmurs were heard, but a presystolic gallop and a paradoxical splitting of the second sound were present. The abdomen was distended and there was questionable shifting dullness. A firm round tender liver edge was palpated 5 cm. below the costal cage. The neurologic examination was within normal limits.

The complete blood cell count, blood sugar and urea nitrogen and serology were normal. The electrocardiogram showed a persistent left bundle branch block, first degree heart block and atrial abnormality. Chest roentgenograms showed right and left chamber enlargement (Fig. 3). Fluoroscopy revealed poor cardiac pulsations. The venous pressure was 130 mm. H_2O and increased to 170 mm. H_2O on hepatic pressure. The circulation time (decholin) was forty-two seconds.

The patient signed out against advice one week later and was readmitted to the Medical Service on October 3, 1958; the physical findings were essentially unchanged. Further laboratory studies included normal liver function tests, protein bound iodine, serum electrolytes and negative L.E. preparations. The patient was given digitalis and diuretics, but still continued to be in respiratory distress. For this reason, extensive pulmonary function tests were carried out and showed the findings associated with cardiac decompensation.

The patient gradually improved with bed rest, but remained dyspneic, with a persistent tachycardia and gallop rhythm. He was discharged after six weeks, but a week later had an exacerbation of his symptoms and was admitted to another institution following a bout of hemoptysis. His condition continued to deteriorate and he died in shock on November 18, 1958.

The clinical diagnosis was idiopathic myocardial hypertrophy.

Fig. 3. Case II. The heart shows the same configuration as in Case I; however, the aortic knob is normal rather than small.

Necropsy. Permission was granted to examine the heart only. The heart weighed 690 gm. and was greatly dilated. The pericardium and epicardium were normal. The myocardium was firm, reddish brown and homogeneous throughout. Histologic section showed hypertrophy and focal interstitial fibrosis. The endocardium was smooth and transparent. No mural thrombi were present. The valves were normal. The coronary arteries were normal in distribution. The walls of these vessels were smooth and showed no occlusions.

The roentgenograms of 8 additional cases with typical findings are shown in Figure 4, *A–J*, and of 4 other cases with nondiagnostic findings in Figure 5, *A–D*.

ROENTGENOGRAPHIC DIFFERENTIAL DIAGNOSIS

Pericardial effusion is the most difficult and important differential diagnosis that must be made.[2,5,10] There have been several cases reported wherein pericardiocenteses were attempted on patients with idiopathic myocardial hypertrophy. Diffuse enlargement of the cardiac shadow with no venous congestion, sharply outlined heart borders, and decreased to absent cardiac pulsations fluoroscopically are common to both idio-

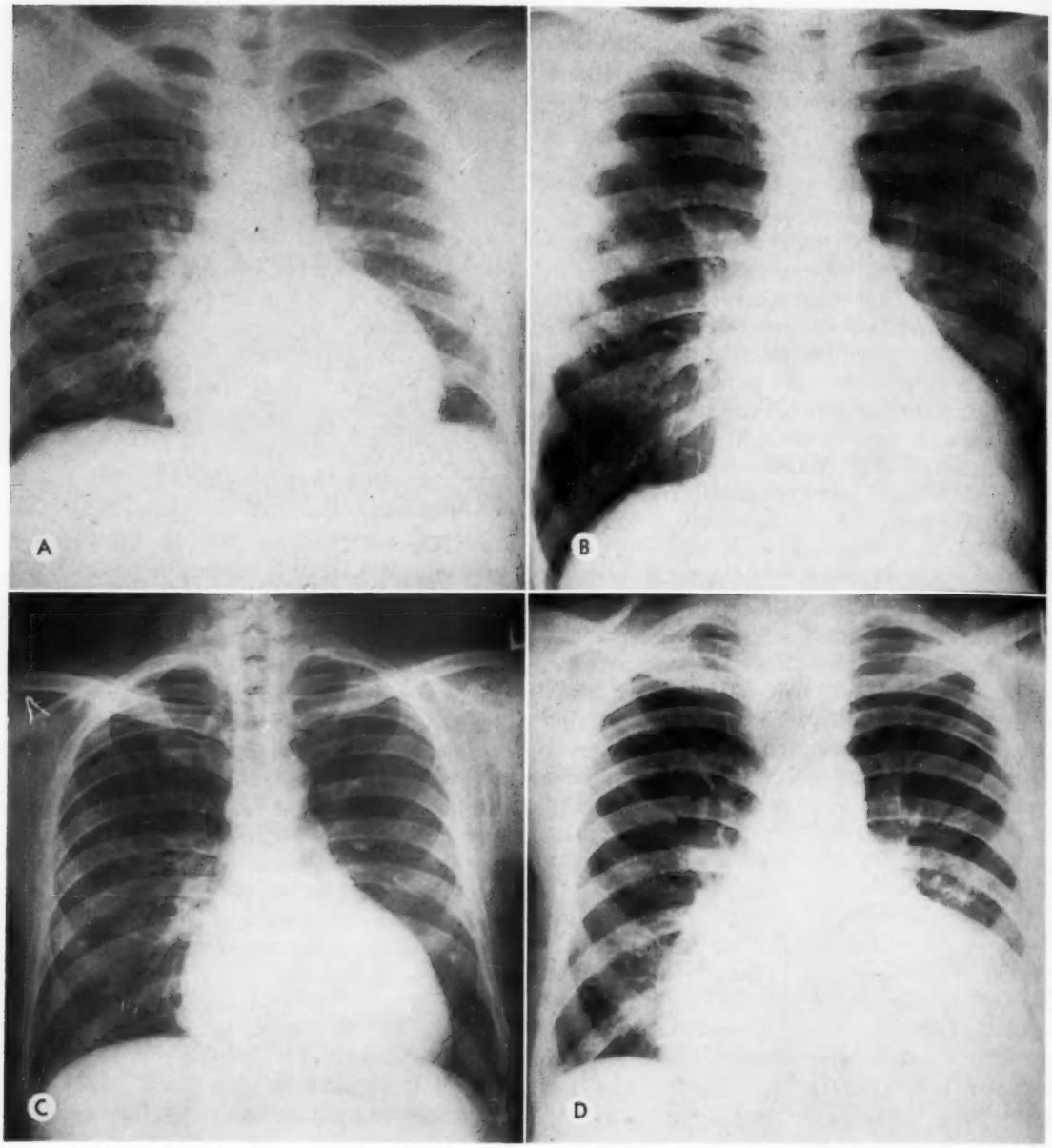

FIG. 4. (*A–D*) These cases show the fairly typical cardiac silhouette seen on the posteroanterior chest roentgenogram. Note the biventricular enlargement with left ventricular predominance. The heart outline is very sharp. Despite the fullness of the pulmonary artery segment, the cardiac waist is still somewhat preserved. The aortic knob is small. The pulmonary vasculature is normal. *C* shows one of the 2 cases with calcification in the aortic knob.

pathic myocardial hypertrophy and pericardial effusion. In pericardial effusion the cardiac waist may be quite broad and there is frequently a loss of the angle made by the shadow of the descending aorta crossing behind the left heart border (Fig. 6). In moderate sized effusions, the normally acute right cardiodiaphragmatic angle becomes obtuse. As the effusion increases, the pericardium bulges laterally beyond its diaphragmatic attachment and the angle again becomes acute. In cardiac dilatation

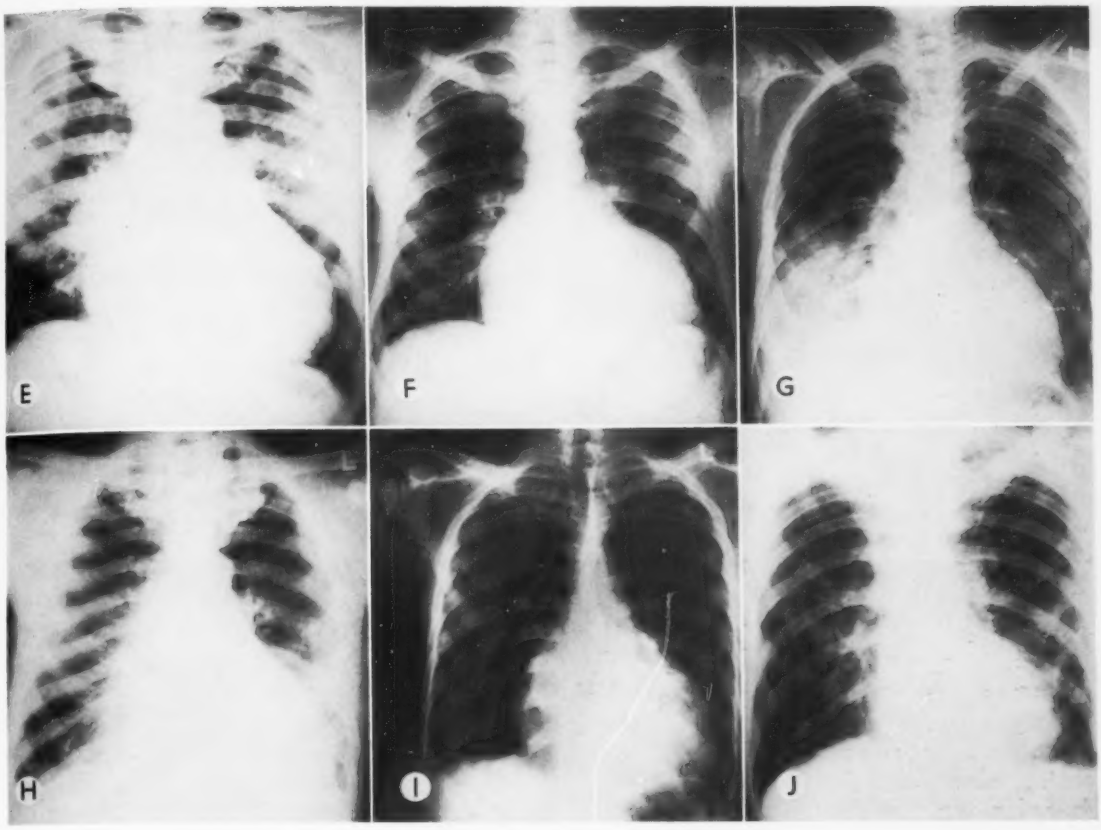

FIG. 4. (*E–J*) Additional cases showing similar changes.

the angle becomes progressively more acute, as it does in large effusions, but it never becomes obtuse. In tuberculous pericardial effusion and myxedema pericardial effusion, the fluid frequently does not extend up to the base of the heart. This may be due to tuberculous adhesions and the viscid character of myxedema effusions. In any effusion where the base of the heart is not involved, it may be impossible to differentiate cardiac dilatation from pericardial effusion on the plain roentgenograms or by fluoroscopic examination (Fig. 7). Where differentiation may be in doubt, angiocardiography is diagnostic. The visualization of a widened pericardial-myocardial shadow between the contrast medium in the right atrium and the outer cardiac border is essentially diagnostic of pericardial effusion.

In pericarditis without effusion the heart is usually small. Pericardial calcification is common in tuberculous pericarditis. Cardiac catheterization reveals a characteristic pressure curve.

Hypertensive cardiovascular disease is probably the most common cause of an enlarged cardiac silhouette in this age group.[5] The diagnosis is usually obvious clinically. However, the "burned out" hypertensive may be clinically indistinguishable from the patient with idiopathic myocardial hypertrophy unless some residual fundoscopic changes are present. Definitive diagnosis is important from the prognostic point of view. The small or relatively small aortic knob of idiopathic myocardial hypertrophy is in marked contrast to the prominent aortic knob seen in hypertensives. In hypertension the left ventricle is the only chamber to enlarge initially. The right ventricle and left atrium enlarge quite

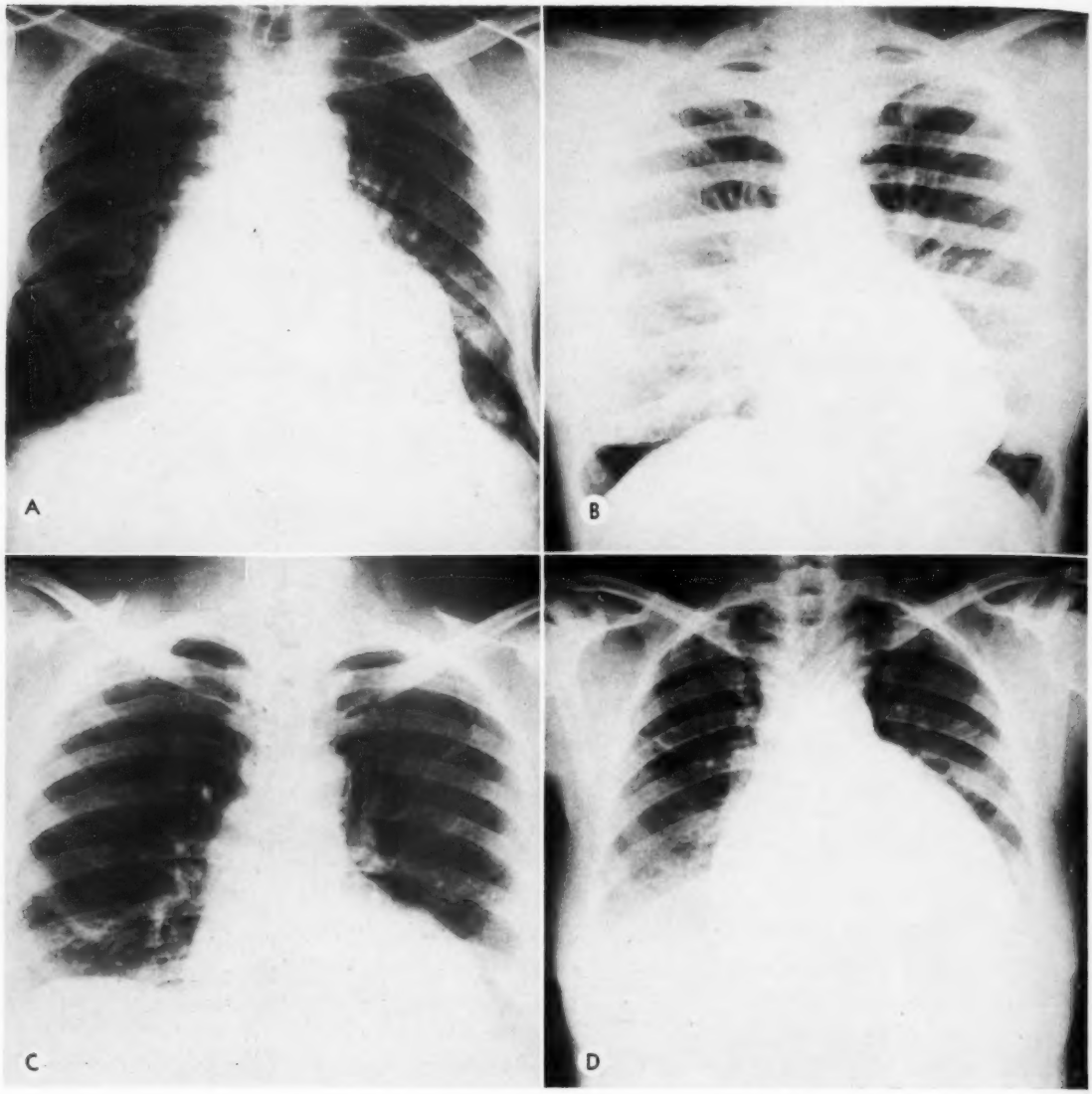

FIG. 5. These are cases with atypical nondiagnostic roentgenographic findings. (*A*) The aortic knob is enlarged in proportion to the left ventricle. (*B*) The right ventricle is larger than usually seen; however, the aortic knob is small. (*C*) This is one of the cases which showed arteriosclerotic changes. Note the aortic calcification. (*D*) The right ventricle is usually prominent. The aortic knob is normal.

late in the course of the disease (Fig. 8). Fluoroscopically the hypertensive heart will exhibit normal or increased pulsations until late when the dilated and weakened heart shows decreased pulsation.

Myocarditis may give a roentgen picture indistinguishable from idiopathic myocardial hypertrophy. However, these patients are febrile and toxic. Electrocardiographic changes may help separate these entities.[9]

Collagen vascular diseases may cause enlargement of the cardiac silhouette. Lupus erythematosus does so by pericardial effusion, and the cardiac findings are no different from those of other effusions. Scleroderma is the only other significant differential diagnosis in this group. The heart is commonly enlarged. Pulsations may be diminished due to collagenous involvement of the myocardium. The pulmonary artery segment is prominent in

patients with extensive pulmonary fibrosis or bronchiectasis.[6] There is usually involvement of many organ systems. The widespread stigmata of scleroderma are in such contrast to the local heart disease of idiopathic myocardial hypertrophy that the differential diagnosis is only rarely a problem.

Cardiac amyloidosis may also present with repeated bouts of congestive failure, frequently of an intractable nature. It usually recurs at an older age group (fifty–sixty years). These hearts are rarely as large as the hearts of idiopathic myocardial hypertrophy.[2] Atrial arrythmias are frequent.[2] Gum biopsies and serum protein analysis may help in the differentiation.

DISCUSSION

Idiopathic myocardial hypertrophy is an eventually fatal disease of unknown etiology occurring in young adults. It is far more common than the few reports in the literature indicate. Discussions with colleagues at other institutions reveal that this entity is being recognized with increasing frequency.

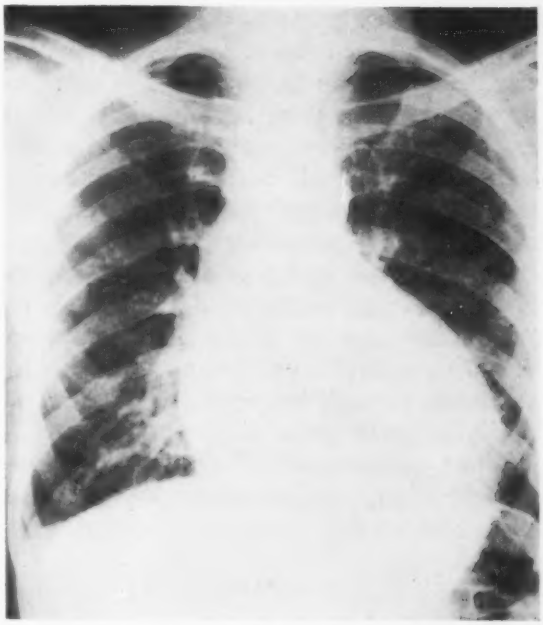

FIG. 7. Tuberculous pericardial effusion. The base of the heart is frequently spared due to pericardial adhesions preventing the movement of fluid. The cardiac configuration is identical with that seen in idiopathic myocardial hypertrophy.

FIG. 6. Pericardial effusion. The cardiac shadow is enlarged and sharply outlined. The enlargement is diffuse. Note how the pericardial fluid has obliterated the normally sharp angle made by the shadow of the descending aorta crossing the left heart border. The right cardiophrenic angle is less acute than one would expect in cardiac dilatation.

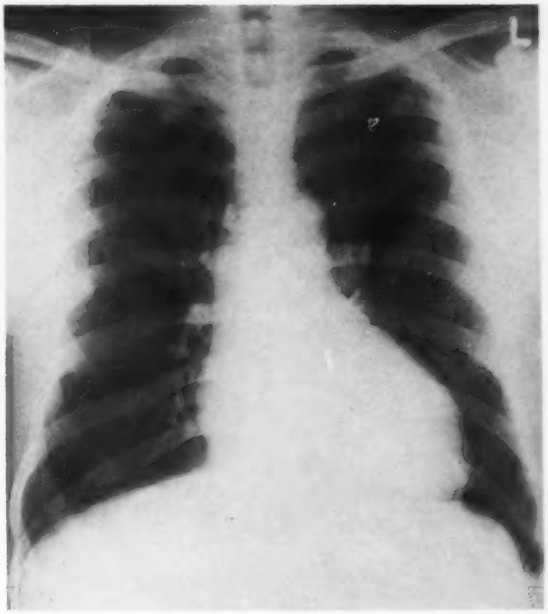

FIG. 8. Hypertensive cardiovascular disease. The left ventricle is enlarged selectively. The aortic knob is prominent and is in proportion to the enlarged left ventricle.

The roentgenographic picture is that of a sharply outlined heart with diffuse ventricular dilatation (left ventricular enlargement predominating), mild left atrial enlargement, occasional right atrial enlargement, a small aorta and somewhat prominent major pulmonary arteries. The frequency of pulmonary and systemic embolization is such that the combination of a cardiac silhouette as described above and pulmonary or mesenteric infarction occurring in a relatively young adult should make one strongly suspect idiopathic myocardial hypertrophy.

When pericardial effusion is a prime consideration, angiocardiography can be diagnostic.

SUMMARY

The clinical, pathologic and roentgenographic findings of idiopathic myocardial hypertrophy are presented with emphasis on the roentgenographic aspects. Two typical cases are included.

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We would like to thank Drs. Mary Ellen Johnson, William Kanenson, Truman G. Schnabel, Jr., Alfred Spivak and Lyle Sensenbrenner for allowing us to report the case histories in detail. We would also like to thank Drs. Samuel Bellet, Morton Robinson and David H. Lewis for their help in the study of these cases.

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Vol. 86, No. 6

STAPHYLOCOCCAL PNEUMONIA IN ADULTS*

A REVIEW OF 102 CASES

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BECAUSE of the paucity and variance of reported cases (a total of 88 cases in four of the largest series in the literature)[3,5,6,9] of a serious disease of rapidly increasing frequency, the cases of staphylococcal pneumonia in adults seen at Wadsworth Veterans Administration Hospital during a seven year period (1953–1959) were reviewed. Table I compares our findings with those in four other adult series and in one large series composed of children. The 102 cases reported here represent approximately half of the total cases admitted; the remainder could not be reported because of the loss or transfer of charts and roentgenograms.

MATERIAL

All patients were male, ranging from eighteen to eighty-two years of age (79 per cent were fifty-five years or older). Hemolytic coagulase positive staphylococci were

TABLE I

MORTALITY AND CALCULATIONS OF STAPHYLOCOCCAL PNEUMONIA

Author	No. of Cases	Mortality (per cent)	Lung Abscess (per cent)	Empyema (per cent)	Pneumatocele (per cent)	Effusion (per cent)	Pneumothorax (per cent)	Bronchopleural fistula (per cent)	Atelectasis (per) cent)
In Adults									
Present Series	102 (1953–1959)	60.5	24.5	7.8	1	37.2	0	0	21.6
Ede *et al.*[3]	36 (1956–1958)	2.8	8	27.7	3.6	22	25	25†	NA
Fisher *et al.*[5]	21 (1942–1957)	67	43*	19	NA	NA	NA	NA	NA
Gold and Davis[6]	13 (1957–1958)	15.4	23	0	NA	23	NA	NA	NA
Hausmann and Karlish[9]	18 (1952–1954)	0	27	11	NA	NA	5.5	5.5	NA
In Children									
Rebhan and Edwards[15]	329 (1950–1958)	14.3	21	8.8	NA	NA	41	3.7	NA
	(Present on admission)		(7)	(NA)	(6.2)	(55)	(21)	(0)	(15.2)

* 10 cases came to postmortems; 9 had lung abscess.
† Reported as pyopneumothorax.
NA—Figures not available.

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cultured in all patients from the following sources: sputum, 90 per cent; blood, 23.5 per cent; pleural effusion, 6.8 per cent; bronchi, 6.8 per cent; lung abscess, 4.9 per cent. Cases of both primary pneumonia (83 cases) and secondary pneumonia (15 cases), *i.e.*, those with previous or coexisting staphylococcal infection elsewhere in the body, were included in the series, with 4 classified as uncertain as to type. The marginal punch card system of recording data was used.

INCIDENCE

Prior to 1950 very few cases of adult staphylococcal pneumonia unassociated with an influenza epidemic were reported. In more recent literature, cases of staphylococcal pneumonia in infants are legion; however, the authors reporting on adult staphylococcal pneumonia infer that this is a relatively rare disease. In recent years there has been a rapidly increasing incidence. In our series, 52 per cent of the cases occurred in 1959.

PREDISPOSING FACTORS

Gresham and Gleeson-White[7,8] feel that alveolar edema may be the common denominator in a host of conditions that predispose a person to staphylococcal pneumonia: chronic lung or heart disease, coma, influenza, suppression of the cough reflex postoperatively, and old age and debilitation. Others agree that staphylococcal pneumonia is a major problem in patients with a pre-existing complicating disease, and that when it does occur in these patients it is more likely to be fatal. This seems to be true, indeed, as 88 per cent of our cases had complicating diseases and the mortality rate was 65.5 per cent, whereas in uncomplicated cases the mortality rate was 25 per cent. Of those with complicating disease, 64.5 per cent had chronic lung or cardiovascular disease and 22 per cent had malignancies. Other complications of lesser frequency were: cirrhosis, diabetes, coma, uremia from chronic renal disease, blood dyscrasias, and general debility. Only those cases with a significant titer were considered to have had influenza; of these there were 2. The *Staphylococcus* as an opportunist invader now has the role previously played by the pneumococci in terminal infection.

Similarly, those patients who have recently received antibiotics or steroids prior to the development of pneumonia, as well as those patients who develop staphylococcal pneumonia while in the hospital (*i.e.*, acquire a hospital organism), are generally agreed to be more prone to develop a more serious and fatal form of the disease. The mortality rate was 72.5 per cent in 29 patients who had recent prior treatment with antibiotics, and 83 per cent in 6 patients who developed staphylococcal pneumonia while on steroids. The majority of our cases (55 per cent) developed pneumonia in the hospital. The mortality rate in this group was 71.5 per cent as compared to 53.5 per cent in that group which presented with pneumonia on admission. When pneumonia develops in a hospitalized patient who is on steroids or antibiotics, it should be considered staphylococcal until proved otherwise.

CLINICAL FEATURES

The clinical pattern of staphylococcal diseases is apparently changing in all age groups, although staphylococcal pneumonia is a more protean disease in adults than in children, who still present a rather characteristic picture. Gresham[8] reports one extreme when he describes a different clinical and pathologic picture which is being seen in adult patients: sudden onset, rapid deterioration, a short course, and hemorrhagic consolidation, or a course similar to the classic description by Chickering and Park[2] of patients developing staphylococcal pneumonia as a complication of influenza. On the other hand, others describe a disease of insidious onset, prolonged course, and a high incidence of complications. Figures 1 and 2 show the type of onset, duration, and mortality for the present series. Although no striking

characteristic clinical pattern was noted in reviewing our cases, the fulminating form of pneumonia was more frequent in the last two years of the study as compared to the earlier years.

The radiologist should be aware of the variable nature of this disease regarding the roentgen findings; they may be present prior to clinical onset or may be completely lacking or minimal, appearing several days later in a severely ill patient. He should be especially conscious of the fulminating type of this disease in which the clinical history of sudden onset and rapid deterioration may lead to the false diagnosis of pulmonary embolism, particularly in the postoperative patient, who not infrequently develops this complication.[11] (Sixteen cases in this series developed staphylococcal pneumonia within a fifteen day postoperative period.)

ROENTGEN FINDINGS

Roentgen examination quite frequently suggests the diagnosis of staphylococcal pneumonia in children because of the extensive and characteristic findings, particularly pneumatoceles and pneumothorax, present on admission. (See Table 1 for Rebhan and Edwards'[15] findings in children.) Adults, on the other hand, usually present with a nonspecific pneumonic infiltration which, in the majority of cases, becomes a bronchopneumonia indistinguishable from any other type of bronchopneumonia, except for the rapidly changing roentgen findings (Fig. 3, *A* and *B*) and the development of pleural effusion, which occurs much more commonly (37.2 per cent) than in streptococcal or pneumococcal pneumonia. In this present series, 98 per cent of the cases had bronchopneumonic infiltrates which were usually bilateral (68.5 per cent) (Fig. 4) and rarely segmental (4 per cent) in distribution. Regarding the amount of lung tissue involved, 59.5 per cent had diffuse involvement.

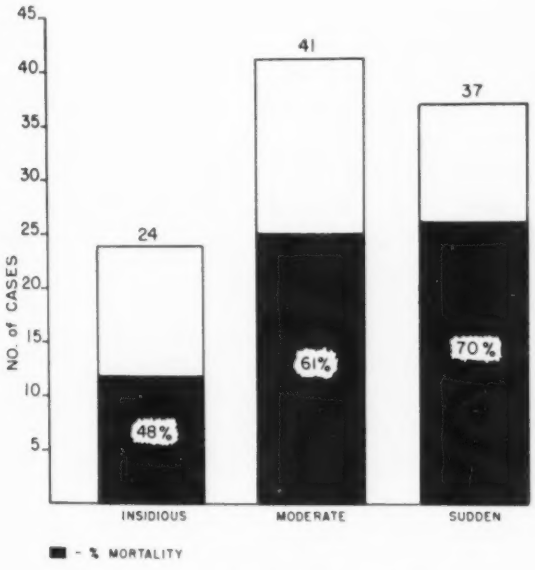

Fig. 1. Type of onset and mortality.

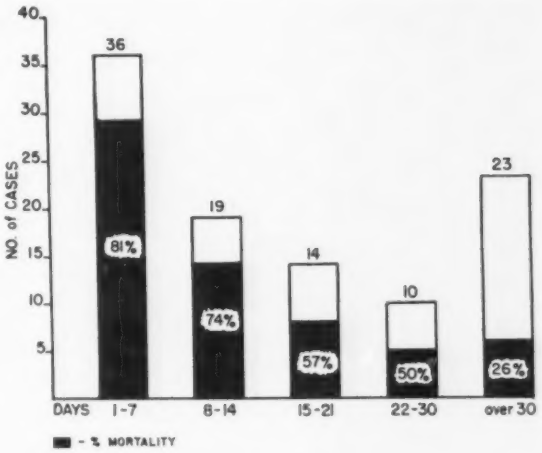

Fig. 2. Duration and mortality.

Frequent roentgen studies are necessary in following this disease because of the rapid change with new areas of involvement (Fig. 3, *A* and *B*). This was seen in 54 per cent of our cases at some time during the course.

The patient should not be discharged until his chest roentgenogram is completely clear. Six patients in this series who were clinically well were discharged before the chest roentgenogram showed complete resolution; all were readmitted with exacerbation of pneumonia within one to four weeks.

The development of pneumatoceles and

FIG. 3. Rapidly changing roentgen findings. (*A*) March 4, 1957: left upper lobe involvement; (*B*) March 6, 1957: moderate clearing on the left with the development of right upper lobe involvement within two days.

pneumothorax, so characteristic of staphylococcal pneumonia in infants, is rare indeed in adults. Only 1 case had pneumatocele formation and there were no cases of pneumothorax. Because of the lack of distinctive features, except those mentioned above, the radiologist's most important function is the early recognition of complications most common in adults—lung abscess and empyema—and following their course.

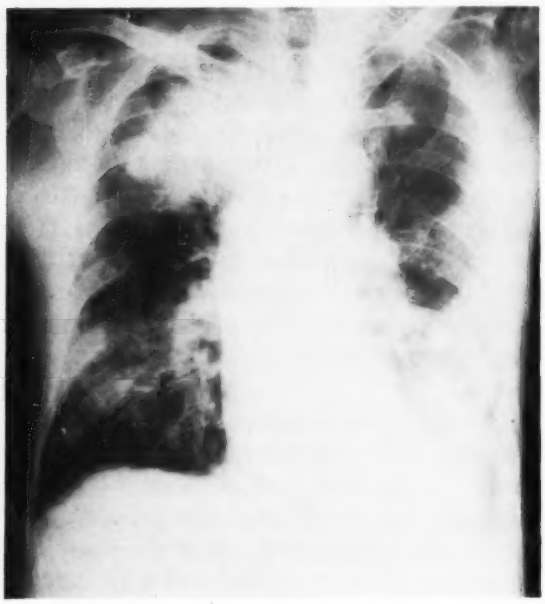

FIG. 4. Typical roentgen findings. October 12, 1957: bilateral bronchopneumonia with multiple sites of involvement.

LUNG ABSCESS

The ability of the *Staphylococcus* to create lung abscesses is primarily due to its exotoxins, which apparently have a dual action: (1) production of vasculitis and paralytic dilatation of capillaries and venules, and (2) a direct necrotizing effect on lung parenchyma.[10]

Meyers and Jacobson[13] state that "true lung abscesses occur more frequently in adults than in children." A comparison of this present series with that of Rebhan's and Edwards[15] indicates only a slightly higher incidence (3.5 per cent) in adults.

Ede *et al.*[3] report that "infiltrates which persist will usually demonstrate cavity formation within ninety-six hours." Rapid

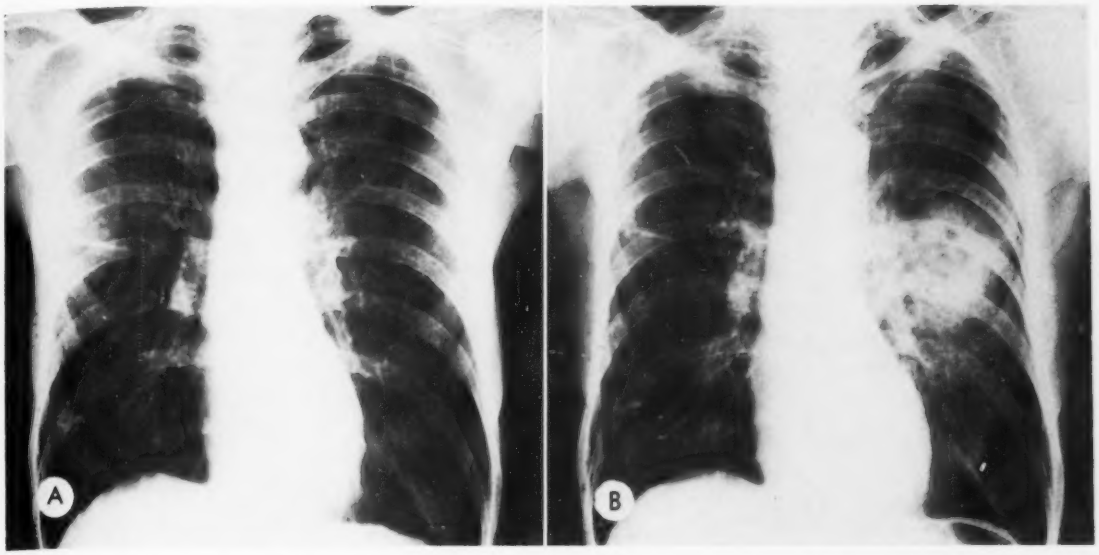

FIG. 5. Rapid development of cavitation. (*A*) October 23, 1959: left lung clear; (*B*) October 26, 1959: multiple cavitations developing in three days.

cavity formation (Fig. 5, *A* and *B*) occurred in approximately one third of our 25 cases of lung abscess (Fig. 6). However, this complication also appeared in some patients after they had had pneumonia roentgenographically demonstrated for over a month (Fig. 7, *A–D*). Figure 8 indicates the roentgenologic duration of cavities once they appeared, usually at least four to six weeks in those patients surviving.

Brock[1] states that the posterior segment of the right upper lobe is the area most frequently involved in staphylococcal lung abscesses. Our random distribution did not bear this out. In cases of secondary pneumonia, *i.e.*, vascular embolism, there seems to be a predilection for the lower lobes, possibly due to the straighter course of the blood vessels. In addition, in cases of both primary and secondary pneumonia, multiple cavities were the rule. Bronchogenic spread undoubtedly accounts for the multiplicity. The same patient may have all phases of development and healing going on at the same time.

The characteristic "soap bubble" appearance of staphylococcal cavities described by Brock[1] was not encountered in our series. By far the greatest number of our cases (72 per cent) had thick and sometimes shaggy cavity walls.

Not only morbidity but also mortality (71 per cent) was higher in patients developing this complication. Indeed, the patient's life is in jeopardy until the lung abscess is roentgenologically demonstrated as being completely healed.

EMPYEMA

Empyema, the second most common complication (7.8 per cent) in our series,

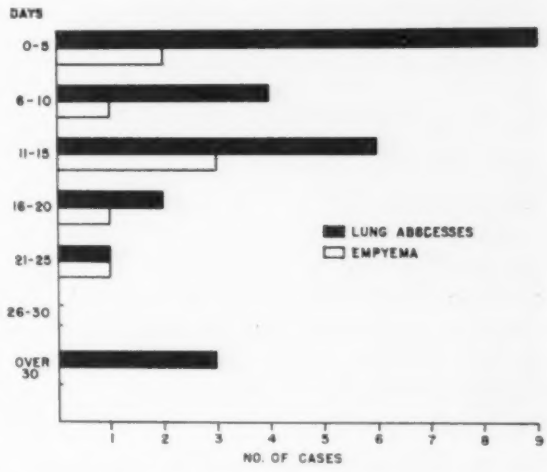

FIG. 6. Length of time between the onset of pneumonia and the development of complications.

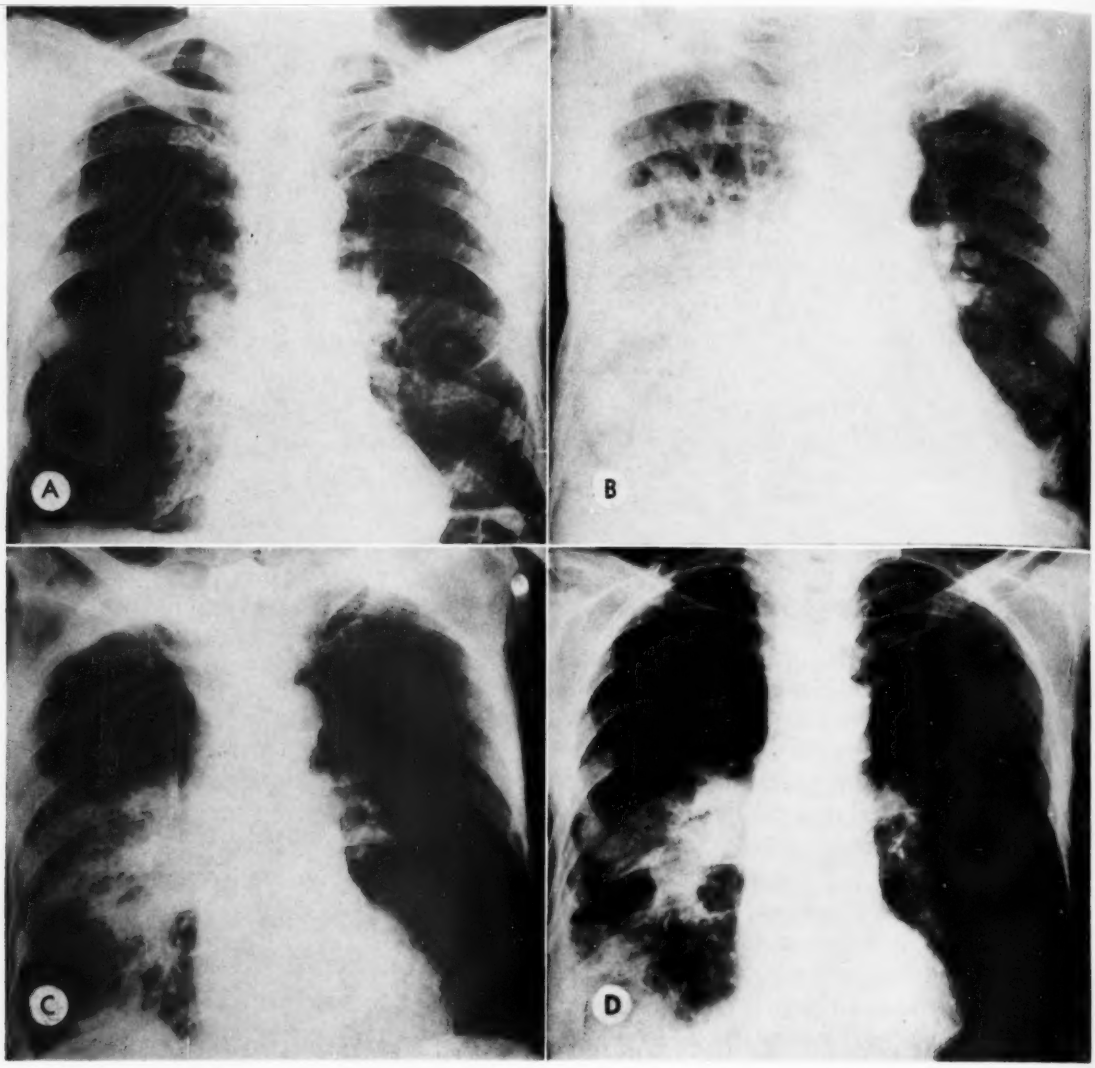

FIG. 7. Late development of cavitation. (*A*) February 8, 1959: minimal findings on the right; (*B*) February 11, 1959: rapid consolidation; (*C*) March 23, 1959: persisting infiltration; (*D*) April 14, 1959: late cavitation after five weeks.

may develop by rupture of a lung abscess, direct extension from the lung via the lymphatics, septic embolization, or contamination during thoracentesis for pleural effusion. Instances of all except the first (rupture of an abscess) were seen in our cases.

Figure 6 indicates the length of time between the onset of pneumonia and the development of empyema; Figure 8 indicates the roentgenologic duration; and Figure 9, *A* and *B* shows the characteristic roentgen findings. Although the length of hospitalization and the morbidity are appalling, this appears to be in adults a less serious complication than lung abscess with regard to mortality (25 per cent). In children, on the other hand, empyema is much more serious, with a mortality rate of 50 to 70 per cent.

All of our cases of empyema were treated by surgical drainage. Before effective drainage can be instituted, exact roentgen localization is necessary. This can be done in

most instances by plain roentgenography though, in low lying empyemas intimately associated with the diaphragm, injection of the empyema with contrast material prior to surgical drainage is important in order to avoid inadvertent puncture of the diaphragm with resultant subphrenic abscess.

In those cases where plain roentgenography is sufficient for localization, once drainage is established, the volume, extent and shape of the affected area can be determined by injecting aqueous dionosil or other suitable contrast media (Fig. 10, *A* and *B*). The volumetric studies thereafter can be performed with sterile saline solution, but the initial examination must be done using a contrast medium and with the patient examined in the erect and decubitus positions to rule out a bronchopleural fistula (Fig. 10 *C*) which, if present, would cause the lung to be flooded if saline solution were injected blindly.[12] Roentgen studies with contrast material are repeated as necessary during the follow-up for two reasons: to rule out the possibility of the development of (1) a bronchopleural fistula, since the empyema will not heal as long as air is supplied to the space, thereby preventing lung expansion, and (2) a blocked off area which, of course, would not drain because of lack of communication.

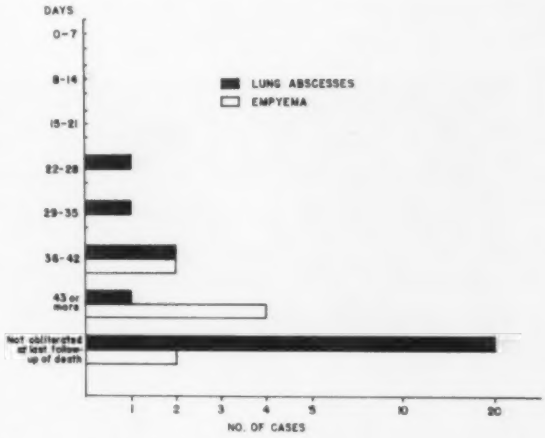

FIG. 8. Roentgenologic duration of complications.

The early roentgen diagnosis of complicating empyema will result in faster healing, since surgical drainage can be instituted before pleural thickening occurs and while the lung can still expand easily.

SUMMARY

A series of 102 cases of staphylococcal pneumonia in adults is reviewed and com-

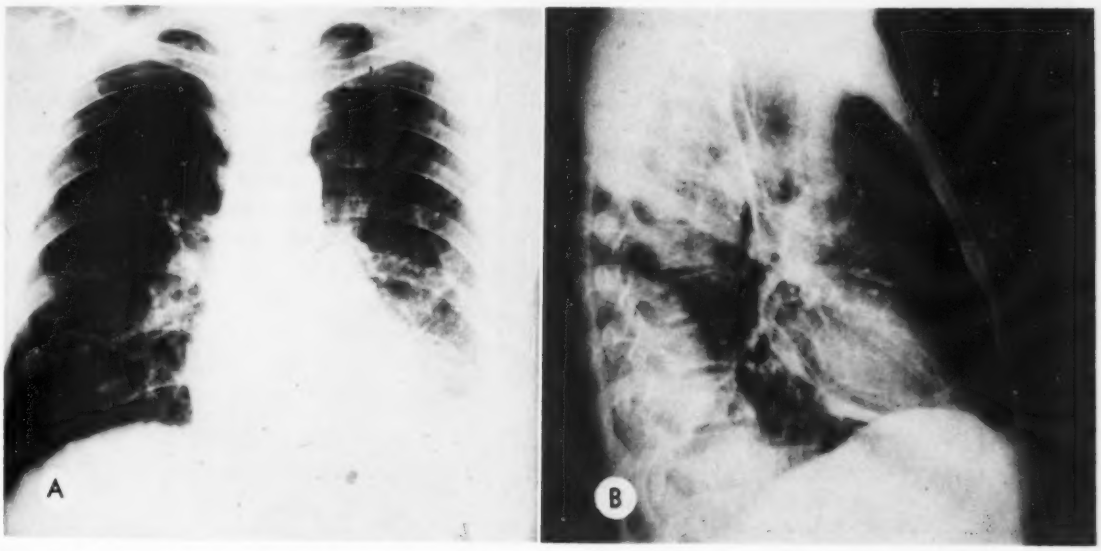

FIG. 9. A case of empyema which developed within one week after the onset of pneumonia and persisted for twelve weeks. (*A* and *B*) December 26, 1957: roentgenograms made midway in the course of the illness show the characteristic roentgen findings.

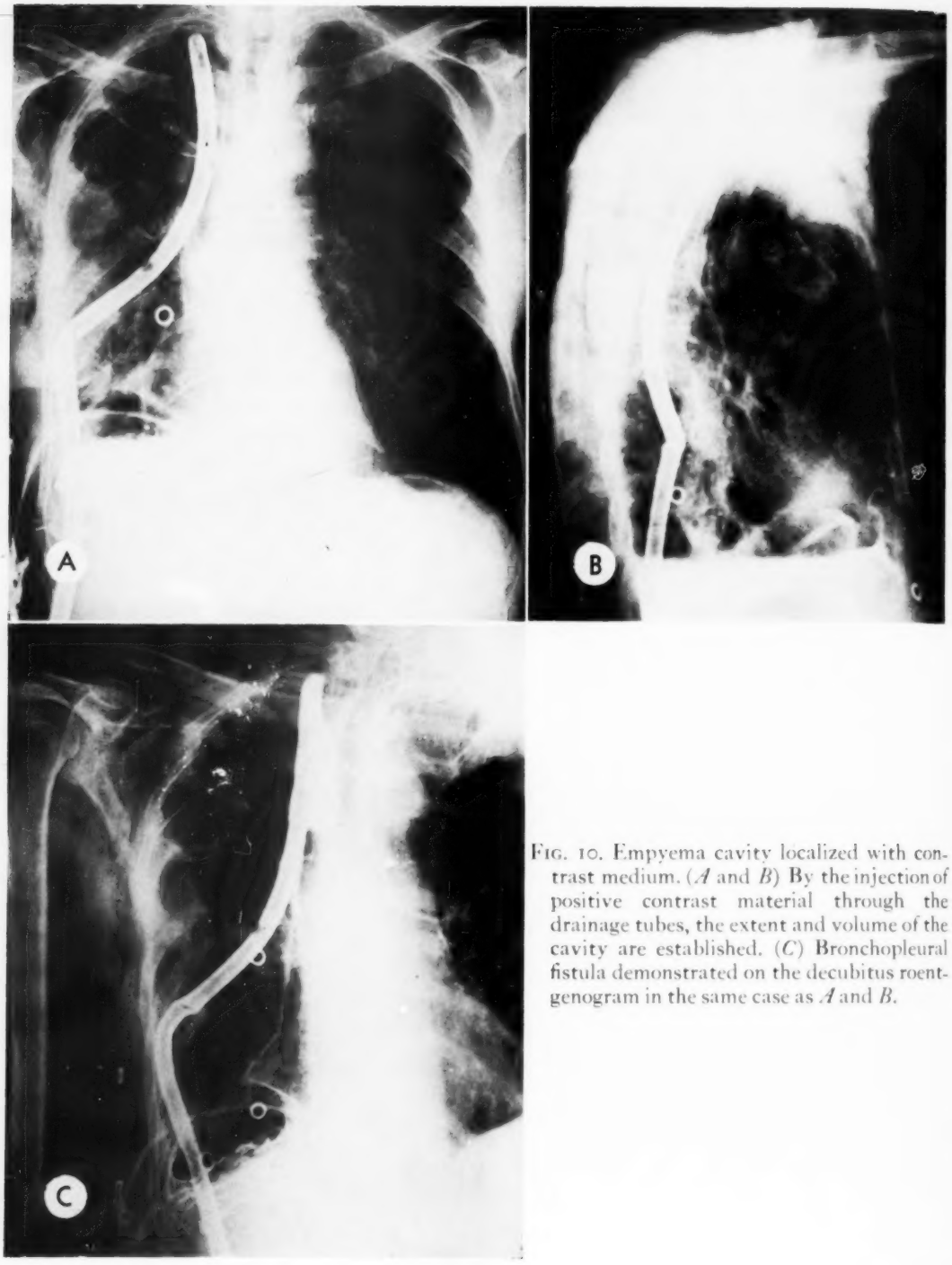

FIG. 10. Empyema cavity localized with contrast medium. (*A* and *B*) By the injection of positive contrast material through the drainage tubes, the extent and volume of the cavity are established. (*C*) Bronchopleural fistula demonstrated on the decubitus roentgenogram in the same case as *A* and *B*.

pared with four other series in adults and one large series in children. The increased incidence, protean nature, and clinical pattern of this disease are described. Because of the relatively nonspecific early roentgen findings, the radiologist's role in the early recognition, localization, and follow-up of complications cannot be too highly stressed.

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SPONTANEOUS PNEUMOTHORAX AS A COMPLICATION OF PULMONARY METASTASES IN MALIGNANT TUMORS OF CHILDHOOD*

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MANY malignant neoplasms of childhood metastasize to the lungs, a variety of secondary effects resulting from their presence. Such manifestations as bloody pleural effusion and compression atelectasis are common, but the development of spontaneous pneumothorax is not often encountered. De Barrin[4] was the first to report the occurrence of spontaneous pneumothorax in a patient with metastatic tumor in the lungs. Including the case reported by him, there have been 29 instances of this complication recorded in the literature.[1–8,10–17] Thirteen additional such patients have been seen at The Children's Hospital and the Children's Cancer Research Foundation (Table I).

The primary tumors implicated in the cases heretofore reported were: osteogenic sarcoma, 9 patients; Ewing's tumor, 5 patients; "carcinoma" (type not specified), 3 patients; leiomyosarcoma, adenocarcinoma, and fibrosarcoma, 2 patients each; renal cell carcinoma, adrenal carcinoma, angiosarcoma, rhabdomyosarcoma, Wilms' tumor (adult type), and carcinoma "à cellules acidophiles," 1 patient each. Of these 29 patients, 7 of the 8 who were sixteen years of age or younger on whom sufficient information was supplied were found to have primary tumors in bone (Table II). The experience at this institution has been similar. Nine of the 13 patients with spontaneous pneumothorax had malignant neoplasms of bone.

In order to investigate this apparent association further, the records of the X-ray Therapy section were reviewed. The

TABLE I

CHILDREN WITH SPONTANEOUS PNEUMOTHORAX COMPLICATING PULMONARY METASTASES
AUTHORS' SERIES

Case		Sex	Age (yr.)	Type of Tumor	Site of Primary Tumor	Site of Pneumothorax
1	R.B.	M	16	Osteogenic sarcoma	Left femur	Right
2	B.H.	F	14	Osteogenic sarcoma	Left femur	Bilateral
3	J.P.	M	13	Osteogenic sarcoma	Left tibia	Bilateral
4	M.E.	F	13	Osteogenic sarcoma	Right tibia	Left
5	R.M.	M	11	Osteogenic sarcoma	Right femur	Right
6	B.S.	M	10	Osteogenic sarcoma	Right humerus	Left
7	L.F.	F	9	Osteogenic sarcoma	Left ilium	Bilateral
8	S.B.	F	10	Ewing's tumor	Left fibula	Right
9	M.O.	F	9	Ewing's tumor	Left humerus	Right
10	M.N.	F	6	Wilms' tumor	Left kidney	Left
11	T.S.	M	5	Wilms' tumor	Left kidney	Right
12	M.DeM.	F	5	Wilms' tumor	Left kidney	Left
13	M.C.	F	11	Hemangioendotheliosarcoma	Left thigh	Right

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This investigation was supported in parts by a research grant from the National Institutes of Health, USPHS #CY3335.
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TABLE II

CHILDREN WITH SPONTANEOUS PNEUMOTHORAX COMPLICATING PULMONARY METASTASES
CASES FROM THE LITERATURE

Authors	Sex	Age (yr.)	Type of Tumor	Site of Primary Tumor	Site of Pneumothorax
De Barrin[4] (1937)	M	15	Fibroblastic sarcoma	Left pubic bone	Left
Christmann *et al.*[2] (1950)	M	12	Ewing's tumor	Left calcaneus	Right
Leigh and Thompson[7] (1951)	M	11	Osteogenic sarcoma	Right tibia	Right
Sherman and Brant[13] (1954)					
Case 1	M	9	Ewing's tumor	Sacrum	Bilateral
Case 2	M	16	Ewing's tumor	Sacrum	Left
Case 3	F	9	Osteosarcoma	Left tibia	Right
Engle[6] (1958)	F	15	Osteogenic sarcoma	Femur	Bilateral
Torricelli and Canossi[15] (1958)	F	11	"Ewing's tumor"	Soft tissues of left shoulder	Left

ten year period 1948–1957 was selected for detailed study. During this interval, 381 patients less than seventeen years of age with malignant neoplasms received roentgen treatment. Some patients with cancer seen at this hospital are not included in the total since roentgen therapy was not required during the course of their illness. Also not represented are those children with lymphosarcoma in whom acute leukemia became manifest. An additional 121 patients (59 boys and 62 girls) had central nervous system tumors. This group has not been analyzed further since no pulmonary metastases developed in any patient.

The roentgenographic findings of each child were reviewed with special attention to the demonstration of pulmonary metastases and an associated spontaneous pneumothorax. No pneumothorax was considered to be spontaneous if preceded by a thoracentesis on the same side. The results of this analysis, according to diagnosis, are given in Table III. When referring to the table, it should be noted that absence of pulmonary metastases does not necessarily imply eventual survival of the patient. In fact, some of the children in this category died from the effects of local or disseminated disease.

A total of 7 cases of spontaneous pneumothorax associated with pulmonary metastases was encountered in the period under study. This represents 2 per cent of the entire group of 381 children, or 6 per cent of the 125 who developed metastases. Five of these 7 patients had primary bone tumors. Osseous neoplasms account for only 13 per cent of the 381 children and 28 per cent of the patients with pulmonary metastases. This compares with but one child with pneumothorax among the patients with Wilms' tumor, which neoplasm comprises 24 per cent of the total and 35 per cent of those with lung deposits. Children with diagnoses other than those already listed made up 63 per cent of the total and 37 per cent of those with metastases. Only one instance of spontaneous pneumothorax was found in this group.

Since osteogenic sarcoma was the neoplasm encountered most frequently in this connection, all children with this diagnosis who developed pulmonary lesions were studied in detail. The points considered were: age; sex; site of primary tumor; distribution of the pulmonary metastases according to location within the inner, middle or outer third of the lung fields; and the osteoblastic, nonosteoblastic or mixed nature of these deposits. The interval between the onset of disease, the development of pulmonary lesions and the time of death was also investigated. No correlative factor was ascertained; the patients with pneumothorax were similar to those who died

TABLE III

ROENTGENOGRAPHIC INCIDENCE OF PULMONARY METASTASES AND OF PNEUMOTHORAX SELECTED GROUP OF 381 CHILDREN WITH MALIGNANT TUMORS

Type of Tumor	No. of Cases		No. of Cases with Lung Metastases		No. of Cases with Pneumothorax			
						Site		
	Total	Sex	Total	Sex	Sex	Right	Left	Bilateral
Neuroblastoma	107	M 60 F 47	11	M 7 F 4				
Wilms' tumor	92	M 45	44	M 20	M			
		F 47		F 24	F		1 (Case 10)	
Bone Tumors Osteogenic sarcoma	27	M 12	19	M 8	M	2 (Cases 1, 5)	1 (Case 6)	
		F 15		F 11	F			1 (Case 7)
Ewing's tumor	23	M 14	15	M 9	M			
		F 9		F 6	F	1 (Case 8)		
Reticulum cell sarcoma	1	M 0 F 1	0					
Rhabdomyosarcoma	14	M 11 F 3	2	M 1 F 1				
Teratoma	8	M 4 F 4	5	M 2 F 3				
Hemangioendothelio-sarcoma	7	M 3	5	M 3	M			
		F 4		F 2	F	1 (Case 13)		
Mixed mesodermal tumor	6	M 5 F 1	2	M 2 F 0				
Angiosarcoma	5	M 1 F 4	2	M 0 F 2				
Adenocarcinoma	4	M 3 F 1	0					
Undifferentiated carcinoma	4	M 3 F 1	3	M 3 F 0				
Neurofibrosarcoma	3	M 1 F 2	3	M 1 F 2				
Undifferentiated sarcoma	3	M 2 F 1	1	M 0 F 1				

TABLE III—(*Continued*)

Type of Tumor	No. of Cases		No. of Cases with Lung Metastases		No. of Cases with Pneumothorax			
	Total	Sex	Total	Sex	Sex	Site		
						Right	Left	Bilateral
Fibrosarcoma	2	M 2 F 0	2	M 2 F 0				
Chondrosarcoma	1	M 1 F 0	1	M 1 F 0				
Miscellaneous carcinomas	14	M 10 F 4	5	M 5 F 0				
Lymphoma and Allied Diseases Lymphosarcoma	19	M 11 F 8	1	M 1 F 0				
Hodgkin's disease	18	M 12 F 6	4	M 2 F 2				
Malignant lymphoma	14	M 13 F 1	0					
Reticulum cell sarcoma (nonosseous)	9	M 4 F 5	0					
Totals	381	M 217 F 164	125	M 67 F 58		4	2	1

with pulmonary metastases but without developing free air in the pleural cavity. However, the impression was gained that in children with osteogenic sarcoma, the first demonstrable pulmonary metastases are frequently located more peripherally than those originating from nonosseous neoplasms. In fact, small subpleural deposits were often the first visible evidence of lung involvement. This distribution may be important in explaining the genesis of pneumothorax in these patients.

Thornton and Bigelow[14] described a young adult with osteogenic sarcoma and spontaneous pneumothorax in whom a bronchopleural fistula was demonstrated at autopsy. The communication resulted from necrosis of a peripheral pulmonary nodule. Lodmell and Capps[8] encountered a patient who exhibited a similar process. In case 3 of our series, two bronchopleural fistulae were found at operation. They extended through friable tumor to the subpleural level where blebs then formed. These in turn ruptured into the pleural cavity (Fig. 1). While there is no doubt regarding the origin of the pneumothorax in this patient, the evidence obtained at autopsy in other children proved more difficult to interpret. Of the 11 patients of this series who died, postmortem examinations were performed in 7. Necrotic neoplastic tissue pouting into the pleural cavity was found in all. Rents could be seen extending deeply into some tumor nodules. These findings could be interpreted as evidence of antemortem

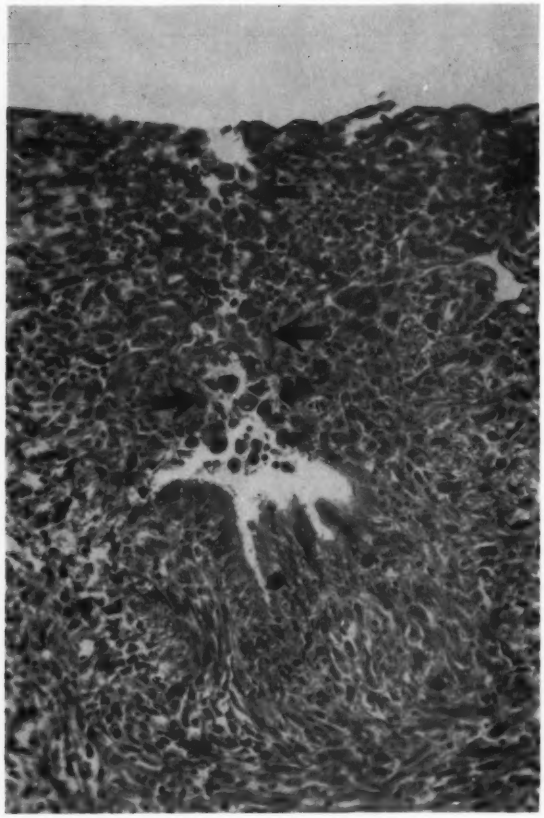

FIG. 1. Case 3. Metastatic osteogenic sarcoma tumor nodule in the periphery of the left lung (×250). The normal parenchyma is almost entirely replaced by masses of neoplastic cells. A fistula (arrows) extends from a bronchiole through friable tumor to the surface of the lung. (Air entered the pleural cavity via a ruptured subpleural bleb.)

bronchopleural communication and thus explain the genesis of pneumothorax. However, dense fibrous adhesions had made removal of the lungs difficult in most of these patients, and often the neoplastic tissue was very fragile and easily traumatized (Fig. 2). It is possible that the changes were artefactual and had been created at the autopsy table. In fact, rents and fissures in necrotic tumor nodules are seen not uncommonly in patients who never developed pneumothorax during life. It is therefore extremely difficult to positively establish the existence of an antemortem bronchopleural fistula, despite careful postmortem examination.

Lodmell and Capps[8] found interstitial air and subpleural blebs in 2 of their patients. They believe that tumor nodules act as ball valves to produce partial bronchiolar obstruction and hyperinflation of alveoli, thus initiating the mechanism described by Macklin,[9] who attributes the development of interstitial emphysema to the overdistention and break-down of alveolar "non-partitional bases" with subsequent dissection of air along perivascular routes to the surface. Should the resulting subpleural blebs rupture, a pneumothorax results. Pulmonary interstitial emphysema leading to subpleural bullae was not identified before or after death in any child in this series. However, the autopsy findings

FIG. 2. Case 6. Anterior surface of the left lung (postmortem specimen). Dense adhesions obliterated much of the pleural cavity, and the lung was torn from its bed in removal. As a consequence, the rough and irregularly thickened pleural surface is disrupted in several areas. The trauma of removal also accounts for the deep fissure visible in the nodule with a denuded surface seen in the right upper corner of the figure. Many other metastases can be identified above the pleural level.

in another child were suggestive. Multiple 3–4 mm. blebs of air were found over the anterior aspect of the mediastinum of this boy who had an osteogenic sarcoma and pulmonary metastases but who never developed pneumothorax.

The evidence derived from the 13 children here reported suggests that the development of bronchopleural fistulae is the more frequent way by which air reaches the pleural cavity. The communications result from either direct extension through the pleura or after the formation of blebs which in turn rupture.

The clinical picture of spontaneous pneumothorax developing in association with pulmonary metastases shows many variations. Some of these are exemplified by reports of cases from the authors' series (Table 1) which follow.

REPORT OF CASES

Case 3. J.P. This male patient, with onset of disease at the age of fourteen, twisted his left knee while playing and developed a painful limp. A roentgenogram showed a lesion in the proximal left fibula and a biopsy was performed. The diagnosis was osteogenic sarcoma. He was then referred to The Children's Medical Center. Roentgen therapy was started, and 8,044 r were delivered to the left proximal tibia in 52 treatments over sixty-three days. Antitumor chemotherapy was also given during this interval. He remained well until four months after the onset of disease when pulmonary metastases were discovered on the right. Three weeks thereafter, roentgen therapy of the entire chest was started and 1,400 r were given in 10 treatments over fourteen days. After an initial diminution in size, the deposits became larger and more numerous. Six weeks following completion of therapy, a silent right pneumothorax with 30 to 40 per cent collapse of the underlying lung was identified roentgenographically. Spontaneous episodes of improvement occurred over the ensuing four weeks, although bouts of coughing became increasingly troublesome. No functional impairment was apparent despite roentgen evidence of persistent residual free air in the pleural cavity. Suddenly, severe coughing spells and pain in the left shoulder became manifest, and another roentgenogram revealed a tension pneumothorax on the left. There had been no definite prior roentgen evidence of left pulmonary involvement even as recently as nine days before. He was admitted to the hospital for observation and treatment. During the next three weeks, repeated left thoracenteses were performed with withdrawal of air, and tubes were inserted twice in an attempt to re-expand the lung. All these measures failed. In view of the persistent and uncontrollable bilateral pulmonary collapse, a left thoracotomy was done in order to identify and close the leaks. A subpleural emphysematous bleb measuring 3 cm. in diameter was found in the superior segment of the lower lobe. Air escaped through a round hole, about 0.5 cm. in diameter, at the apex of the bleb. No tumor was palpable in relation to this bulla, but microscopic examination revealed a bronchopleural fistula through a tiny 4 mm. metastasis (Fig. 1). Another ruptured bleb in relation to a metastasis, less than 2 cm. in diameter, was found along the pleural surface of the lingula near its juncture with the upper lobe. These lesions and a third subpleural nodule were removed. The surface of the lung was then scarified and dusted with talc, and the chest was closed around two tubes. The left lung reinflated promptly and remained expanded throughout the ensuing five months of life. The pneumothorax on the right followed a variable course, but at no time cleared completely. The patient continued to fail slowly and expired fifteen months after the first signs of disease.

Comment. The uncontrolled tension pneumothorax on the left, added to the right pneumothorax, severely restricted functional capacity. There was also an ever-present danger of complete bilateral collapse. Hospital care was considered essential under the circumstances. All conservative measures failed to secure re-expansion on the left and thoracotomy became necessary to achieve this end. There followed prompt improvement, enabling the child to leave the hospital and remain at home for the remainder of his life.

Case 5. R.M. This male patient, with onset of disease at the age of eleven, was well until he began to complain of pain in the right knee. One month later, swelling of the part had become noticeable and a biopsy of the right distal

femur was performed. The microscopic diagnosis was osteogenic sarcoma, and roentgen treatments were started, approximately 1,200 r depth dose being given in fifteen days. The child was then brought to The Children's Medical Center. Tumor chemotherapy was initiated, and roentgen therapy directed to the distal femur was continued, using 2 mev. technique.* An additional 6,000 r tumor dose was given, making a total of 7,200 r in fifty days. Six months later, roentgenograms showed a pulmonary deposit on the right, and four weeks thereafter, metastases were identified in the left distal femur. Eight months after the development of chest metastases and without prior symptoms referable to their presence, the patient suddenly became disoriented and perspired profusely. This was accompanied by moderate pain in the right chest. The entire episode lasted about twenty minutes. He was admitted to the hospital the next day when a chest roentgenogram showed a massive right-sided tension pneumothorax. The lung slowly re-expanded over the next seven weeks. Chemotherapy with various agents had been continued throughout his course, but the chest metastases and those in the left femur continued to enlarge. The right pneumothorax recurred two weeks after it had been resorbed, and on this occasion a thoracentesis was done with removal of the free air. His status continued to deteriorate rapidly and he died eighteen months after the onset of disease.

CASE 7. L.F. This female patient, with onset of disease at the age of nine, was in good health until she suddenly complained of pain in the left thigh and inability to stand. Two weeks thereafter a mass along the left iliac crest was noted. Roentgenograms showed an extensive osteolytic lesion involving most of the wing of the left ilium. No metastases in the lungs or elsewhere were present at that time. Radiation therapy of the ilium was started, and after a depth dose of 824 r in seven days, a biopsy was performed. The pathologic report was osteogenic sarcoma. The patient was then continued on 2 mev. radiation therapy.* She received an additional 2,290 r (depth dose) in sixteen days and one month later 3,900 r (depth dose) in twenty-nine days. Tumor chemotherapy was then initiated. She remained entirely well with no pulmonary symptoms until two months later, when she developed vomiting and slight pain anteriorly over the chest, which was aggravated by deep inspiration and coughing. Roentgenograms taken within the next few days demonstrated a tension pneumothorax on the right with complete collapse of the lung (Fig. 3*A*). The margins of the right lung showed numerous small metastatic nodules. These were also present on the left side. On the same day, a thoracentesis was performed on the right side and 600 cc. of air was removed. Two days thereafter, however, a tension pneumothorax and complete collapse of the lung were again demonstrated on the same side. Nine days later there supervened nausea, vomiting, and greatly increased respiratory embarrassment. Roentgenograms showed a tension pneumothorax and complete collapse of the lung still present on the right side. A pneumothorax also was present on the left with about 50 per cent collapse. (Fig. 3*B*). Bilateral thoracotomies for constant drainage of both pleural cavities resulted in considerable improvement. The tubes were removed when re-expansion was complete. Ten days later, pneumothorax recurred bilaterally, with more than 50 per cent collapse of the lungs on each side. The tubes were reinserted but only slight improvement resulted. She died one month later of severe pulmonary insufficiency, six months after the onset of disease.

At autopsy, hydropneumothorax was found on both sides. Each lung was collapsed and extensively involved with tumor masses. The visceral pleura appeared to be irregular and disrupted bilaterally due to necrotic tumor nodules and fibrous adhesions to the thoracic cage. Many of the pulmonary metastases in each lung showed necrotic centers.

CASE 8. S.B. This female patient, with onset of disease at the age of ten, developed painless swelling of the left calf which, on roentgen examination, was found to be caused by a tumor in the fibula. This was biopsied and the microscopic diagnosis of Ewing's tumor was made. Roentgen therapy was delivered to the entire left fibula over a thirty day period. The depth dose was calculated to be 6,400 r minimum and 7,100 r maximum. Meanwhile tumor chemotherapy was started. A mid-thigh amputation was then performed. Four months thereafter, pulmonary metastases on the left were visible on roentgenograms. These were treated with

* Cases 5 and 7 were treated through the kind cooperation of Dr. Joseph Marks and associates of the New England Deaconess Hospital.

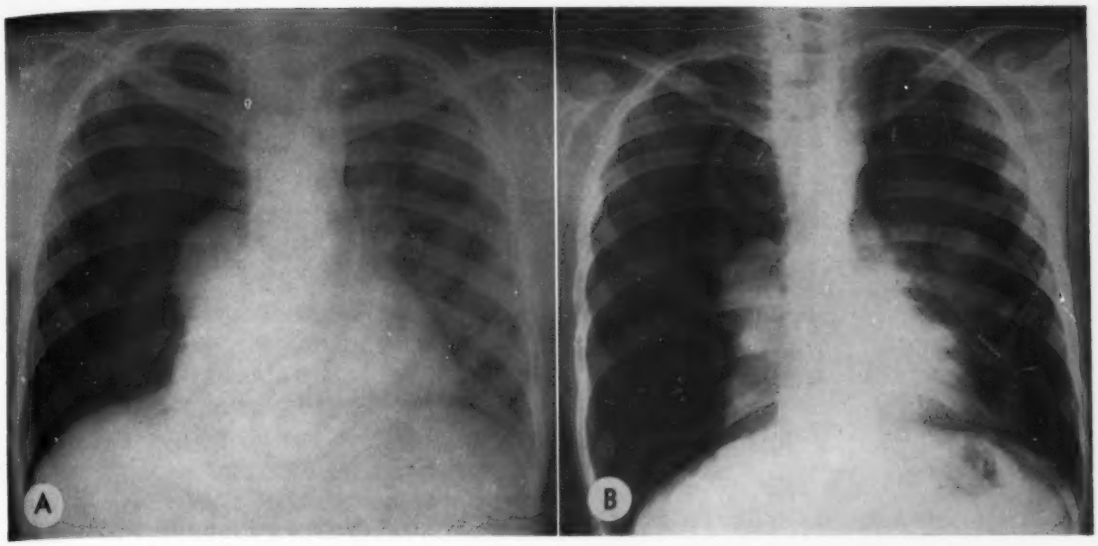

Fig. 3. Case 7. (*A*) Osteogenic sarcoma with bilateral pulmonary metastases. A spontaneous tension pneumothorax on the right is associated with complete collapse of the lung. This complication was the first indication that pulmonary deposits were present. (*B*) Ten days later. Pleural free air on the right persists despite attempts to secure permanent re-expansion. A spontaneous pneumothorax is now present on the left where multiple subpleural metastases are visible.

palliative irradiation with consequent temporary disappearance. Four months later, new lesions were visible on the right. Slow growth ensued over the next seven months, when exertional dyspnea and severe cough developed. A chest roentgenogram revealed the presence of a small, spontaneous tension pneumothorax on the right (Fig. 4). There were no further signs or symptoms attributable to the free air which resorbed during the following four weeks and never recurred. Her course continued to be downhill. The child expired thirty-three months after the onset of illness.

At autopsy, the entire thoracic cage was filled with tumor masses and fibrous adhesions largely obliterated the pleural cavities. Virtually no normal lung tissue could be identified grossly. Many of the neoplastic lesions showed extensive central necrosis.

Case 10. M.N. This female patient, with onset of disease at the age of six years, had been well until a palpable mass in the abdomen on the left was discovered. Excretory urography confirmed the presence of a left renal tumor, and a left nephrectomy was performed. The diagnosis on microscopic examination was Wilms' tumor. No pulmonary metastases were present at this time. Postoperative roentgen therapy of the left renal bed was given through opposing anterior and posterior portals. The depth dose was 3,500 r delivered in 22 treatments in twenty-seven days. Seven months later, a Horner's syndrome developed on the left and metastatic involvement was found in the left cervical lymph nodes. Pulmonary metastases on the right were also discovered at that time. Chemotherapy was started, and the roentgen therapy was directed to the cervical mass and to the pulmonary lesions, but her subsequent course was progressively downhill. Ten months after the nephrectomy, pleural fluid accumulated on the right, requiring thoracentesis for relief of respiratory distress. Two months thereafter, an asymptomatic spontaneous pneumothorax developed on the left. Gradual re-expansion of the lung took place during the remaining four weeks of life. She expired thirteen months after the onset of disease.

At autopsy, there were extensive metastases to the lungs, pleura, and tracheobronchial lymph nodes as well as to various extrathoracic sites. Bilateral hemothorax was also present, with severe compression atelectasis of the right lung.

Case 13. M.C. This female patient, with onset of disease at the age of eleven years, was first admitted to the hospital because of pain and swelling of the right thigh. An incomplete

FIG. 4. Case 8. Ewing's tumor with pulmonary metastases and spontaneous pneumothorax. A peripheral tumor nodule bulges into the pleural cavity.

excision was performed with histologic diagnosis of hemangioendotheliosarcoma. Roentgen therapy was delivered to the right thigh. Pulmonary metastases became evident five months thereafter. Her condition deteriorated slowly despite the administration of multiple chemotherapeutic agents with and without roentgen therapy.

Fifteen months later there was a sudden episode of difficulty in breathing and chest pain which persisted over the ensuing four days. Roentgen examination of the chest showed a large tension pneumothorax on the right with complete collapse of the lung. A drainage tube was inserted into the right chest. Within twenty-four hours there was almost complete re-expansion of the lung. Further episodes of collapse occurred, but the lung completely re-expanded within two weeks. She continued to fail and died twenty-two months after her first symptoms.

At autopsy, there was extensive bilateral pulmonary disease, but no explanation for the genesis of the pneumothorax on the right was established.

DISCUSSION

There appears to be a disproportionate number of children with osseous tumors in whom spontaneous pneumothorax is a complication of pulmonary metastases. Chest roentgenograms demonstrating this combination suggest that the primary tumor originates in bone and most probably is an osteogenic sarcoma. A corollary is that patients with this neoplasm and lung deposits not infrequently develop a spontaneous pneumothorax. Of 19 children with osteogenic sarcoma metastatic to the lung, 4 manifested spontaneous pleural free air at some time during the course of the disease.

The complication can develop relatively early in the course of a child with a malignant process and pulmonary deposits. In some patients, the presence of free air in the pleural cavity is the first sign of lung involvement, previous roentgenograms having failed to demonstrate metastases. The pneumothorax may be silent and unsuspected clinically, only to be discovered as an incidental finding on roentgen examination. No treatment may be necessary under circumstances such as these, particularly since spontaneous re-expansion can occur. While the improvement may be only temporary in some patients, in others there is no recurrence.

The onset of pneumothorax is dramatic at times, and the patient develops the classic signs and symptoms of sudden chest pain, dyspnea and collapse. Prompt recognition and effective management of the complication, when disabling, can result in several more months of comfortable existence for the patient. Conservative measures may suffice, but vigorous treatment often becomes necessary. This may prove difficult, and thoracotomy sometimes is required before permanent re-expansion of the affected lung is obtained.

It has been suggested[8] that cough may be

a precipitating factor in the production of pneumothorax in these patients. Free air can develop, however, without any previous complaint referable to the chest. In Case 7, the onset of tension pneumothorax was the first indication of pulmonary metastases, the child having been free of symptoms with normal roentgenographic findings prior to that time.

No obvious association could be found between any form of chemotherapy and the development of this complication, nor was there any constant relationship to the use of roentgen therapy to the chest.

The roentgen differential diagnosis of spontaneous pneumothorax seldom presents any difficulty in these patients. Occasionally, extensive necrosis within huge pulmonary metastases with tracheobronchial communication can lead to confusion in diagnosis. The resultant air-fluid level may seem to reach the thoracic wall and simulate the appearance of a pneumothorax (Fig. 5). Metastases with associated large subpleural blebs have been described. An adult reported by Lodmell and Capps[8] was found at autopsy to have bullae as large as 20 cm. in diameter. Roentgenographically, these might simulate a localized pneumothorax. A child with metastatic osteogenic sarcoma (not included in this series) presented such a problem in roentgen diagnosis. It was not possible to state with certainty whether there was a small loculated pneumothorax or whether the appearance was caused by a collection of blebs beneath the pleura (Fig. 6).

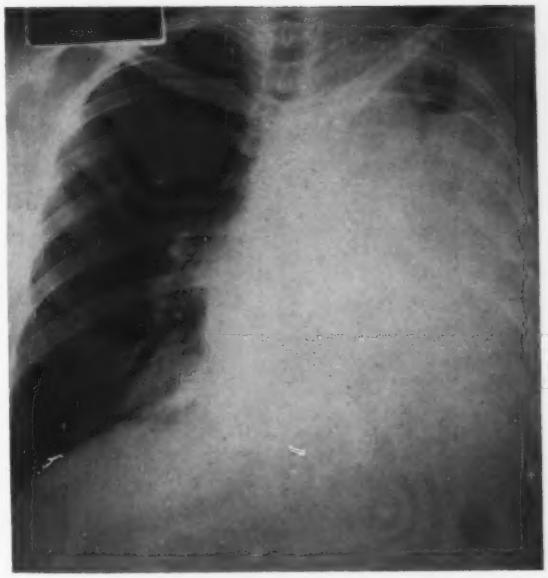

Fig. 5. Osteogenic sarcoma with pulmonary metastases. An air-fluid level is present in the left upper lung field and simulates a hydropneumothorax. At autopsy, no free air was present in the pleural cavity. There was extensive necrosis and liquefaction within an enormous tumor mass which virtually replaced the lung, but a thin shell of intact tissue remained peripherally.

SUMMARY

Spontaneous pneumothorax occurs infrequently in association with pulmonary metastases. Thirteen children with various malignant tumors who developed this complication are reported. Nine of these patients had bone tumors, as did 7 of the 8 children heretofore recorded. The apparent association of this secondary manifestation and malignant tumors primary in bone is discussed as are the probable routes by which free air reaches the pleural cavity.

The onset of pneumothorax may be asymptomatic in some instances but disabling in others. Since the complication may occur early in the course of the disease, prompt detection and treatment can secure for some patients relative comfort during the remaining months of life.

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We are indebted to Drs. Sidney Farber and William T. Green for making available the clinical data of these patients.

ADDENDUM

Since this paper was prepared for publication, an additional case has come to our attention. Dargeon, in his book "Tumors of Childhood" (Paul B. Hoeber, Inc., New York, 1960), reports the case of a nine year old boy with an angiosarcoma,

FIG. 6. Osteogenic sarcoma with pulmonary metastases. A small collection of air is visible immediately beneath the parietal pleura (arrows). The appearance could be produced by a small loculated pneumothorax or by subpleural blebs. At autopsy, two weeks after this roentgenogram was made, extensive adhesions were found in the area, but neither free air nor bullae were identified.

primary in the sacrum, with bilateral pulmonary metastases and bilateral pneumothorax.

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VOL. 86, NO. 6

BLEEDING ESOPHAGEAL VARICES IN THE PRESENCE OF A NORMAL SPLENOPORTOGRAM

A CASE REPORT

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PERCUTANEOUS splenoportography was originally devised to provide anatomic and functional information on the portal venous system to the surgeon planning a shunting procedure. A clinical diagnosis of portal hypertension, supported by routine laboratory and roentgenographic studies, has usually been made beforehand. Recently, refinements in technique, particularly in rapid serial roentgenography, have led to a more widespread use of this examination. At present, splenoportography is no longer restricted to candidates for surgical shunting but is now also advocated by many as a diagnostic procedure in upper gastrointestinal bleeding. In this regard, surprisingly meager data are available in the literature concerning the diagnostic accuracy of splenoportography. In general, however, all investigators have verified the high degree of accuracy that might be anticipated theoretically. Thus, Turner, Sherlock and Steiner[12] consider venography the method of choice in demonstrating esophageal varices and state: "If portal venography fails to show a gastroesophageal collateral circulation, bleeding from varices is most unlikely." Bergstrand and Ekman[1] reported filling of collaterals in all patients with portal hypertension and expressed the opinion that barium swallow studies never demonstrated varices that did not visualize at venography. Rousselot *et al.*[9] state: "We expected and found in the portogram, coronary-gastroesophageal collaterals in *all* cases."

We have recently been distressed by our failure to demonstrate collaterals in a patient with portal hypertension and obvious varices. For this reason, the following case is presented together with some discussion concerning the possible hemodynamic mechanisms involved.

REPORT OF A CASE

This sixty-seven year old white retired railroad worker was referred to the Hospital of St. Raphael because of weakness and hematemesis. The admission diagnosis was bleeding peptic ulcer. Six days before admission, the patient noted tarry stools and vague epigastric discomfort not relieved by meals. He was seen by his private physician and placed on an ulcer regimen of antacids and antispasmodics. One day prior to, and on the day of, admission, he vomited considerable amounts of bright red and coffee ground material.

Six months previously, he had noted black stools over a one week period, associated with weakness and mild epigastric discomfort. On an ulcer regimen, the symptoms subsided and no further studies were done. There was a long history of chronic alcoholism but no previous episodes of jaundice. Traumatic amputation of his left lower leg had resulted from a railroad accident ten years before.

Physical examination revealed an extremely pale but alert and oriented male. No jaundice or spider angiomata were noted. The lungs and heart were normal. A questionable nontender liver edge was felt but the spleen was not palpable. The blood pressure was 130/70 and the pulse 120.

The hemoglobin on admission was 4 gm. and the hematocrit 17 per cent. The bromsulphalein was 20 per cent, but the remaining liver function studies, including total protein, A/G ratio, bilirubin, prothrombin time, cephalin flocculation, thymol turbidity, cholesterol, alkaline phosphatase, and blood ammonia, were normal.

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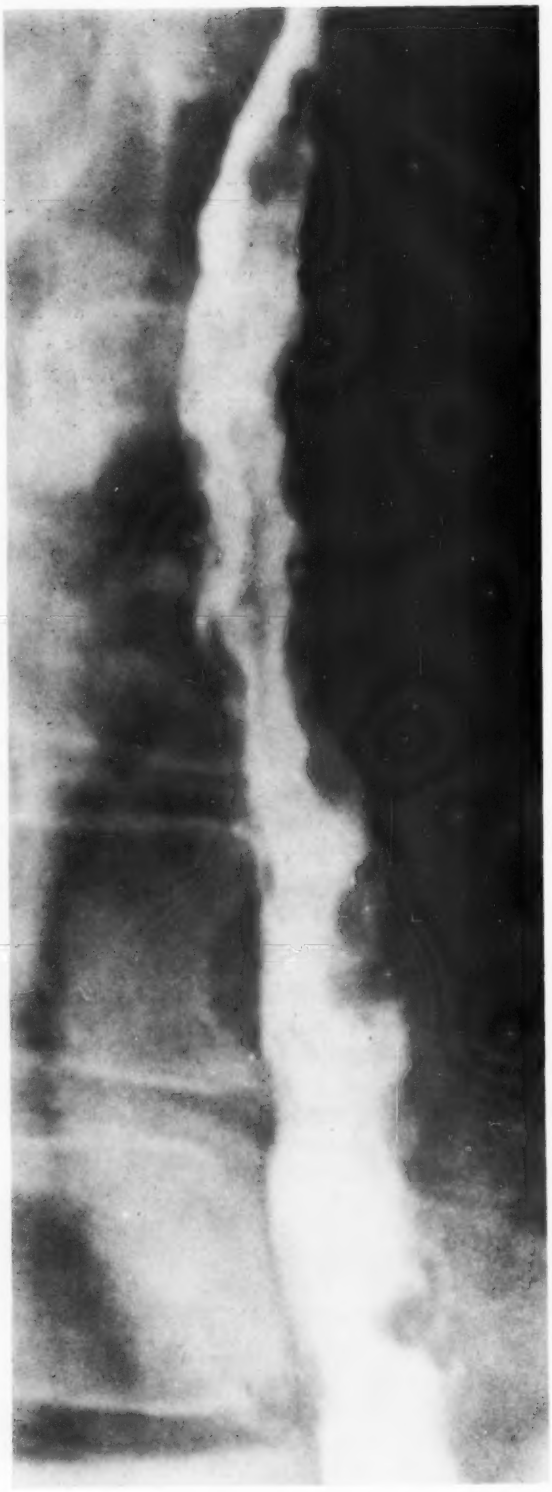

FIG. 1. Barium swallow study demonstrating large varices extending the length of the esophagus.

An upper gastrointestinal series revealed large varices of the distal esophagus and probable gastric varices (Fig. 1).

During the first three hospital days, multiple transfusions raised the hemoglobin to 11.5 gm. and all evidence of active bleeding subsided. In view of the well compensated hepatocellular function, a splenoportal shunt was contemplated. In preparation for this procedure, percutaneous splenoportography was performed. Although not palpably enlarged, the spleen was easily punctured and the intrasplenic pressure (corrected to the anterior body of the first lumbar vertebra[8]) averaged 410 mm. H_2O. Following the manometric determination, 50 cc. of 90 per cent hypaque was injected manually over a period of six to eight seconds. Roentgenograms at the rate of two per second were obtained commencing with the beginning of the injection and extending over a fourteen second period. At no time was any collateral circulation or retrograde filling of the portal tributaries demonstrated. Patent splenic and portal veins were visualized. The intrahepatic portal branches were sparse and tortuous, with the typical "tree in winter"[10] appearance associated with cirrhosis (Fig. 2).

Failure to demonstrate any roentgenographic signs of portal hypertension (*i.e.*, retrograde filling of portal tributaries, visualization of collateral circulation, dilated splenic and portal veins with delayed transit) in the presence of

FIG. 2. The initial splenoportogram without evidence of collateral filling. Note the filling defect at the entrance of the superior mesenteric vein. Intrasplenic pressure at this time was 410 mm. H_2O.

marked intrasplenic hypertension and obvious esophageal varices raised some doubt concerning the correctness of the clinical diagnosis. A localized left coronary vein occlusion can produce varices and a normal splenoportogram, but such a patient would not benefit by portal vein decompression. Carcinoma of the gastric cardia frequently extends submucosally up the esophagus and occasionally may simulate varices.

Consequently, four days after the first splenoportogram, esophagoscopy was performed. Very large varices were encountered 7 cm. below the cricopharyngeus and extended the length of the esophagus. The following day, a second percutaneous splenoportogram was performed, technically similar to the first examination in all details. On this occasion, retrograde filling of the inferior mesenteric vein and visualization of short gastric veins draining into large esophageal varices confirmed the original diagnosis of cirrhosis with portal hypertension and varices (Fig. 3). The corrected intrasplenic pressure on this occasion was 370 mm. H_2O.

An end-to-side portacaval anastomosis was performed (Dr. Philip Wade) resulting in a pressure drop of 110 mm. H_2O (300→190) when the shunt was opened. A repeat barium swallow study during convalescence showed almost complete disappearance of the varices, a few widened longitudinal folds remaining in the distal esophagus.[4]

The patient was admitted five months following surgery in a stuporous condition with a blood ammonia of 105. He responded rapidly to low protein feedings, his sensorium clearing in thirty-six hours and the blood ammonia dropping to 42. Unfortunately, no barium swallow examination was done at this time. However, there has been no recurrence of gastrointestinal bleeding.

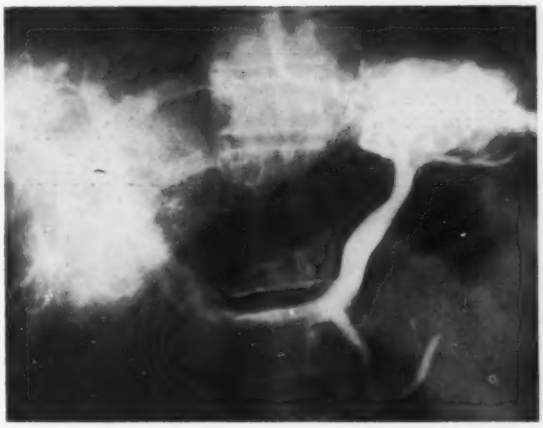

Fig. 3. Note the retrograde filling of the inferior mesenteric vein and the dilated short gastric veins filling tortuous esophageal varices. Intrasplenic pressure was 370 mm. H_2O.

DISCUSSION

Most reviews of splenoportography have stressed its accuracy in demonstrating portal collateral circulation, particularly the cardioesophageal collateral system. We believe that serial roentgenograms are required for an adequate examination and that failure to visualize known varices on a single roentgenogram at the conclusion of injection does not exclude their filling at an earlier or later phase. Bruwer and Hallenbeck[3] include in their material a single case (Case 5) in which splenoportography showed no evidence of collaterals in spite of a portal pressure of 520 mm. H_2O and varices previously demonstrated on barium swallow study. However, only a single roentgenogram was taken and the authors themselves admit that "more than one roentgenogram is required for satisfactory demonstration of the functional hemodynamic status of the portal system." Figley *et al.*[6] mention 3 cases with proved varices which did not fill but they also incriminate inadequate roentgenography. In addition, no mention is made of intrasplenic pressure measurements or the visualization of other collateral systems.

The time sequence of examinations may have an important bearing on collateral filling. It is conceivable that varices, demonstrated on barium swallow examination during acute hepatic decompensation, might fail to fill during splenoportography at a later date. Intensive medical therapy may undoubtedly ameliorate acute hepatic congestion with a resultant increase in transhepatic portal blood flow and a drop in portal pressure. Previously overburdened

TABLE I

Examination	Hospital Day	Collaterals Demonstrated	Portal Pressure
1st gastrointestinal study	2	+	
2nd gastrointestinal study	7	+	
1st splenoportogram	8	−	410 mm. H_2O
esophagoscopy	14	+	
2nd splenoportogram	16	+	370 mm. H_2O

collateral networks may then cease to function or diminish markedly in size. However, this possibility fails to explain the findings in our case (Table 1) in which collateral filling occurred only after clinical improvement and a drop in portal pressure.

The natural history of esophageal varices has been well demonstrated by Palmer,[7] who performed serial esophagoscopies on 133 patients with proved cirrhosis and varices, noting a marked day to day variation in their size and extent. In 35 of the subjects, varices were not seen during at least one esophagoscopic examination. In addition, he found little correlation between varix size and the clinical course of the patient or the level of portal pressure. He explained this variation by pointing out that esophageal varices constitute but one of several portosystemic shunting systems and that different outflow tracts may function at different times. Theoretically, splenoportography should demonstrate all collateral networks functioning at the time of the examination and should, therefore, be more reliable than either a barium swallow study or esophagoscopy where only the submucosal component of the cardioesophageal shunt can be seen. Failure to fill varices at splenoportography, provided other shunt systems are demonstrated, does not exclude their presence. However, failure to demonstrate any collaterals at all in the presence of portal hypertension, as happened in our case, would seem to exclude a large functioning collateral network. Certainly, there must be a stage early in the development of cirrhosis when elevated portal pressure alone suffices to perfuse the liver and no collaterals are present. This was the stage of disease with which we thought we were dealing. If uncertain or equivocal varices had been demonstrated on barium swallow examination, we would have pursued it no further.

From our personal experience and a review of the literature, a small but definite group of patients with portal hypertension and functioning portosystemic shunts seem to have a normal splenoportogram. Steiner *et al.*[11] from a large experience, report 2 patients with varices not demonstrable on splenoportography, but they make no mention of other collaterals. Evans[5] reports 4 cases showing extensive varices on barium swallow studies with apparently normal portograms. However, 2 of these patients had normal intrasplenic pressures and the third, an unsatisfactory pressure tracing. It is conceivable that, with normal portal pressures, none of the shunts was functioning at the time of the examination. (He does not mention the time relationships between barium swallow examination and splenoportography). This leaves but one case similar in all respects to our own, *viz.*, a normal splenoportogram in the presence of portal hypertension and proved collaterals.

It is difficult to conceive of a mechanism to explain this phenomenon. Evans[5] suggests that breath holding during the examination might elevate the pressure in the caval and azygos systems with temporary arrest or even reversal of the flow through collaterals. It is our policy to have the patient breathe shallowly throughout the examination except when the needle is inserted or withdrawn. A prolonged injection time resulting in an inadequate bolus of contrast material should produce faint opacification of all vascular channels, not merely failure of collateral filling. Moreover, Bergstrand and Ekman[2] have shown in dogs that variations in the rate of intrasplenic injection, provided adequate visu-

alization is obtained, produce no variation in the presence or absence of collateral filling. It is possible that streaming phenomena within the portal vein could sweep the entire bolus of contrast material directly to the liver in spite of the presence of multiple functioning shunts.

No adequate hemodynamic explanation may be offered for the above case. Nevertheless, it does caution against complete reliance upon splenoportography for the diagnosis of esophageal varices. Although splenoportography is the most reliable single technique available at present, it is not infallible since it can yield normal findings in the presence of bleeding varices.

SUMMARY

A case of bleeding esophageal varices is presented with two percutaneous splenoportographic studies. The first examination was normal but the second study, performed under similar conditions, revealed the varices. Possible hemodynamic mechanisms are considered.

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INTRAVENOUS ABDOMINAL AORTOGRAPHY IN THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF ANEURYSMS OF THE SPLENIC, HEPATIC AND RENAL ARTERIES*

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THERE is general agreement that the definitive diagnosis of aneurysm of the splenic, hepatic or renal artery is established by contrast visualization. The era of abdominal aortography was ushered in by dos Santos *et al.*[7] in 1929 when they described their lumbar aortography. At first, largely because of the alarming vascular damage and fatalities in the animals studied by Henline and Moore[20] and reported from this center, there was reluctance to perform translumbar aortography. Later, it became quite popular,[8,32,33,47,54,55] but had been virtually abandoned by many as a diagnostic procedure when fatalities and increasing complications were reported.[5,25,28] However, it is still highly regarded in many centers for diagnosis of renal arterial malformations and for discovery of the cause of renal hypertension, and with proper care and precaution can be accomplished with minimum risk.[5,10,35,36] Lately, percutaneous retrograde aortography and selective arteriography have been widely used, probably because of improvements in catheters,[42,43] spring guides, contrast agents, and mechanical injectors.[9]

A recent account of an intravenous method of abdominal aortography, employing a modification of the Robb-Steinberg technique, by bilateral, simultaneous and rapid injection of concentrated organic iodides and precisely timed roentgenography documents the capability of the procedure to readily opacify aneurysms of the aortic arch, abdominal aorta and its branches.[48] Because very little special or expensive apparatus is required, it can easily be performed in the average well-equipped radiology department. Abdominal aortography and peripheral arteriography, thereby, become practical and generally available diagnostic techniques.

In this report, intravenous abdominal aortography for diagnosis of aneurysms of the splenic, hepatic and renal arteries is illustrated, discussed and compared with arterial puncture. In this way, the place of intravenous abdominal aortography in establishing or excluding disease of the abdominal aorta and its branches can be assessed and evaluated.

FINDINGS

ARTERIOSCLEROTIC SPLENIC ARTERY ANEURYSM

Three patients with arteriosclerotic aneurysms of the splenic artery have been examined since we began using intravenous abdominal aortography in 1959. The first case, (Fig. 1, *A* and *B*) recently reported,[49] was studied after a roentgenogram of the hips, made because of backache, had disclosed calcifications in the left upper quadrant (Fig. 1*A*). An intravenous abdominal aortogram showed that the larger of the two calcifications was a splenic artery aneurysm, the other a calcified lymph node (Fig. 1*B*).

Another patient[50] (Fig. 2, *A–D*), a woman aged sixty-seven, with gastrointestinal complaints, was found to have an eggshell type of calcification just medial to the lesser curvature of the stomach (Fig. 2*A*). Intravenous abdominal aortograms demonstrated a splenic artery aneurysm (Fig. 2, *B* and *C*). Figure 2*D* is a view of the aneurysm at operation, while Figure 2*E* is the excised specimen.

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Aided by a grant from the Whitehall Foundation.

The third case, that of a sixty-five year old woman (Fig. 3, *A–D*), was unusual because the presenting complaint was hematuria, which subsequently was found to be due to hemorrhagic cystitis. The conventional roentgenograms showed a 2.5 cm. round calcified mass in the left upper quadrant, seemingly related to the left kidney (Fig. 3, *A* and *B*). Intravenous abdominal aortograms, however, clearly differentiated it from the kidney substance and enabled the establishment of the diagnosis of an arteriosclerotic splenic artery aneurysm (Fig. 3, *C* and *D*). Figure 3*E* shows the splenic artery aneurysm *in situ* at the time of operation. The aneurysm and spleen were removed; the convalescence was uncomplicated.

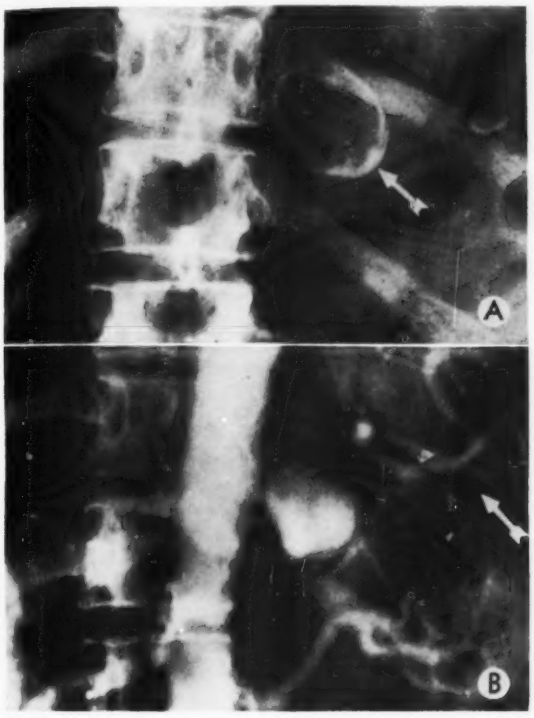

Fig. 1. *Arteriosclerotic aneurysm of the splenic artery in an asymptomatic fifty-four year old housewife.* (*A*) Enlarged (close-up) conventional roentgenogram showing two calcific shadows in the left upper quadrant. The larger lesion is egg-shell in type and located between the eleventh and twelfth ribs adjacent to the eleventh and twelfth vertebrae. The smaller rectangular lesion is in an interspace above. (*B*) Enlarged (close-up) intravenous abdominal aortogram showing the opacified splenic artery aneurysm. Note that the upper calcific shadow (arrow) is not opacified.

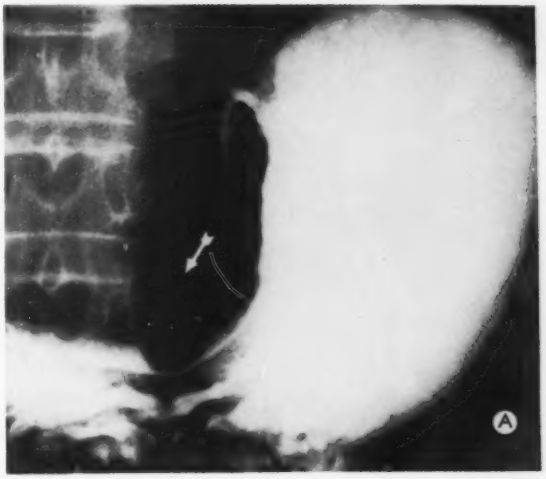

Fig. 2. *Arteriosclerotic splenic artery aneurysm in a sixty-seven year old woman with vague left upper quadrant flatulence and discomfort.* (*A*) Enlarged (close-up) roentgenogram of the barium filled stomach showing a rounded 1.5 cm. egg-shelled type of calcification adjacent to the left border of the spine just below the eleventh left rib (arrow).

HEPATIC ARTERY ANEURYSM

The case of an arteriosclerotic aneurysm of the hepatic artery in a seventy-two year old man (Fig. 4, *A–C*) was also recently reported.[50] The aneurysm, encountered during an exploratory operation for subacute cholecystitis and cholelithiasis, was not removed. Four years later, when we began employing abdominal aortography, the hepatic artery aneurysm was visualized. Figure 4*A* is the conventional roentgenogram of the abdomen showing a small calcification in the right upper quadrant. An intravenous abdominal aortogram showed an aneurysm of the hepatic artery (Fig. 4, *B* and *C*).

RENAL ARTERY ANEURYSM

Two patients with renal artery aneurysms have had intravenous abdominal aortography. The first, a fifty-five year old woman (Fig. 5, *A–C*) had hypertension (190/110 mm. Hg), peripheral vascular disease, and a characteristic signet-ring calcific shadow in the pelvis of the right kid-

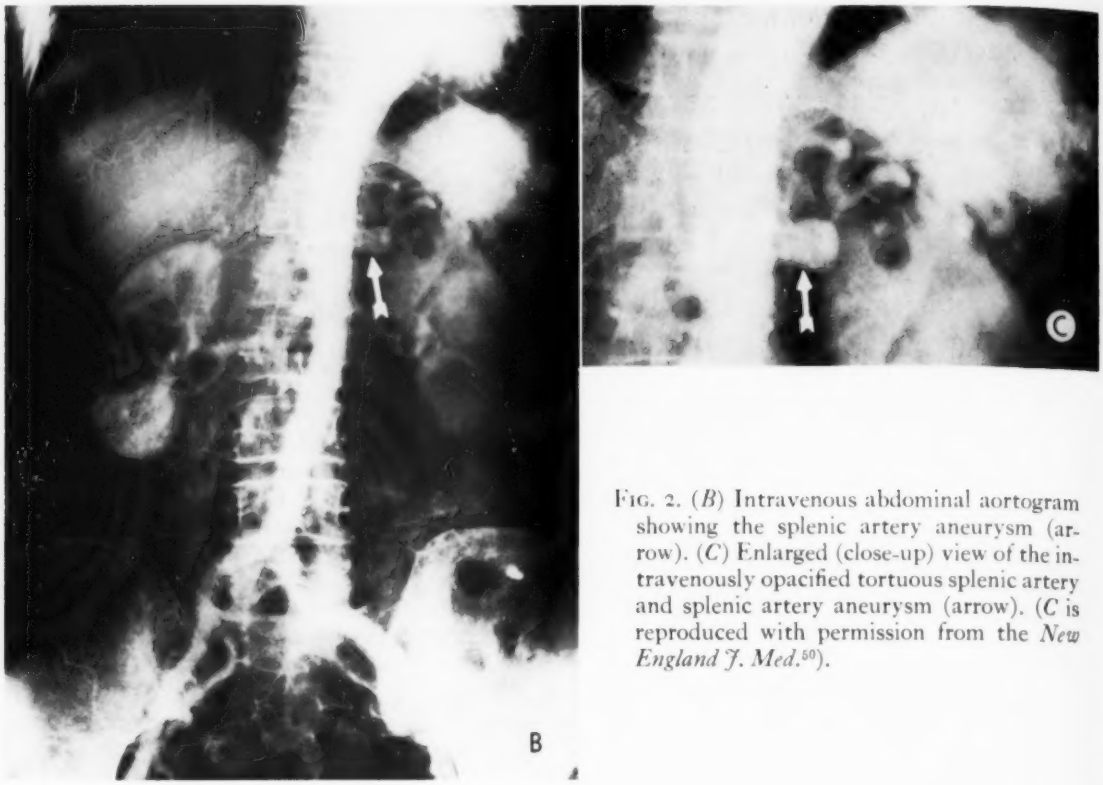

FIG. 2. (*B*) Intravenous abdominal aortogram showing the splenic artery aneurysm (arrow). (*C*) Enlarged (close-up) view of the intravenously opacified tortuous splenic artery and splenic artery aneurysm (arrow). (*C* is reproduced with permission from the *New England J. Med.*[50]).

ney (Fig. 5, *A* and *B*). An intravenous abdominal aortogram demonstrated a hypoplastic left renal artery and kidney, and arteriosclerotic endarteritis of the abdominal aorta and iliofemoral arterial tree (Fig. 5*C*). The left renal artery was poorly visualized, probably because of arteriosclerotic endarteritic involvement of the vessel. The right renal artery was not abnormal; an aneurysm in the hilus of the

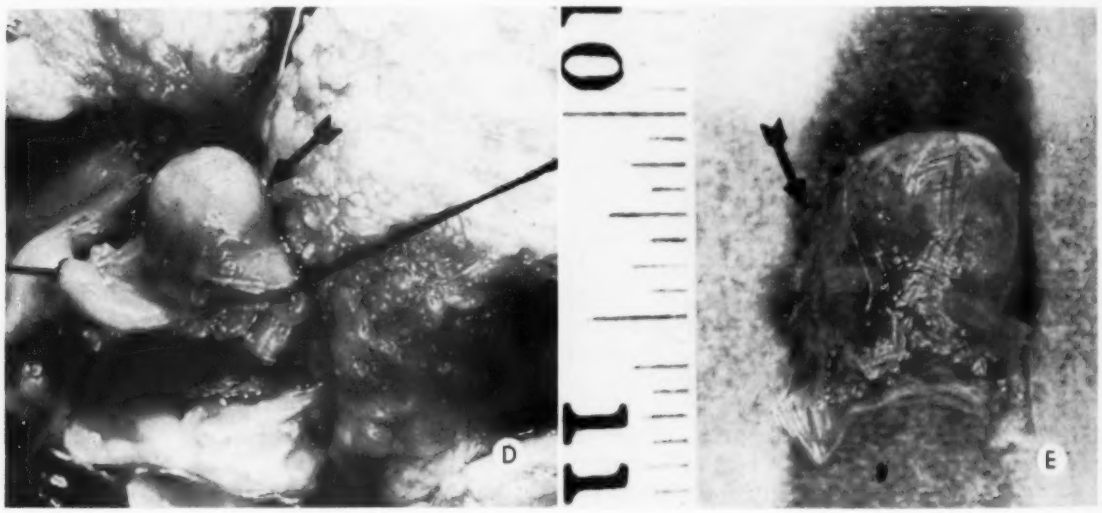

FIG. 2. (*D*) Photograph of the splenic artery aneurysm (arrow) at operation. (*E*) The excised splenic artery aneurysm.

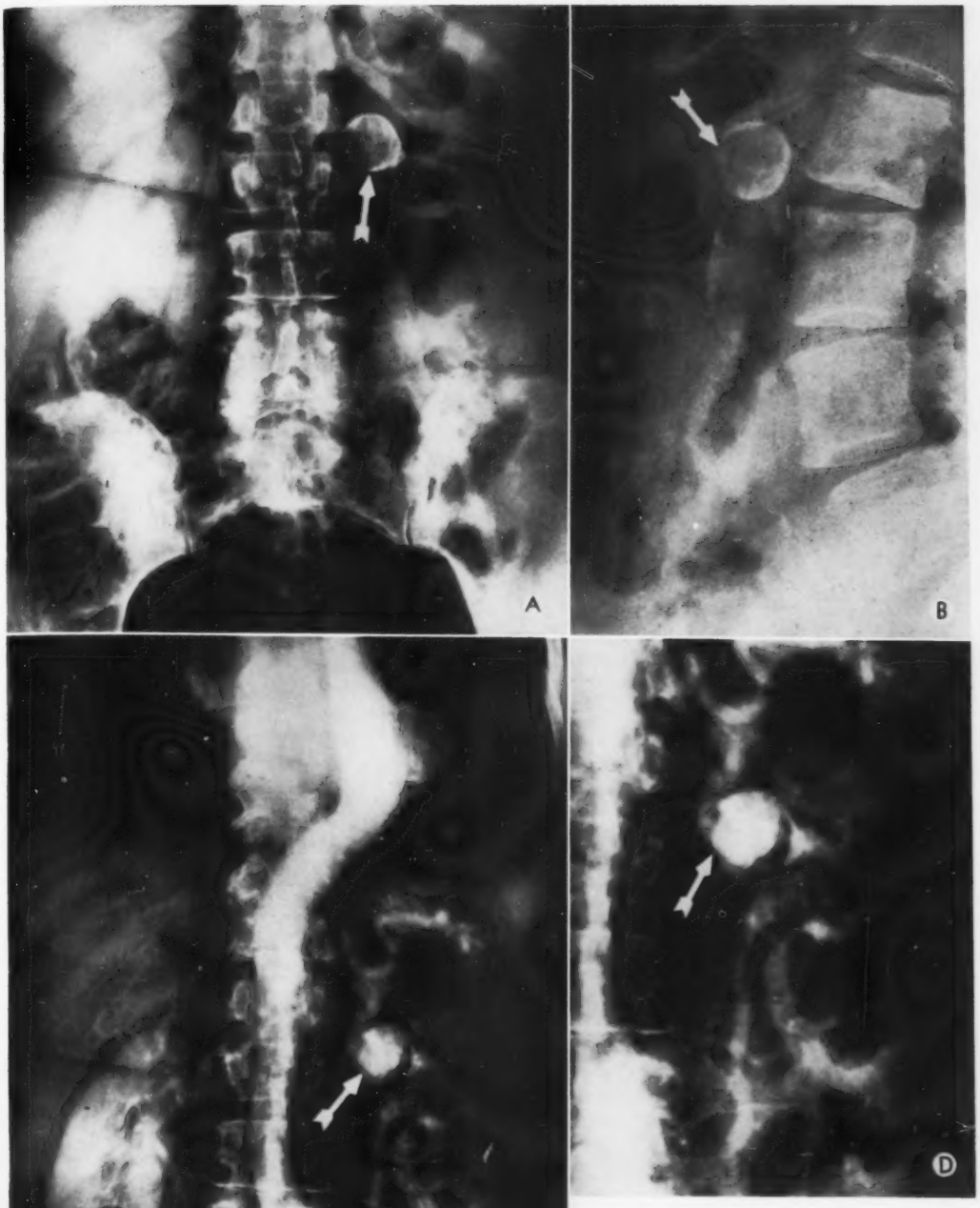

FIG. 3. *Arteriosclerotic splenic artery aneurysm in a sixty-five year old housewife admitted with a complaint of hematuria.* (*A*) Conventional abdominal roentgenogram showing a rounded 2.5 cm. calcific (egg-shell) lesion in the left upper quadrant (arrow) just below the left twelfth rib. (*B*) Lateral roentgenogram of the abdomen showing the location of the calcific lesion (arrow) deep in the left upper quadrant. (*C*) Intravenous abdominal aortogram disclosing the splenic artery aneurysm (arrow) and the tortuous splenic artery. Tortuosity of the abdominal aorta and common iliac arteries is seen. (*D*) Enlarged (close-up) view of the opacified tortuous splenic artery and splenic artery aneurysm (arrow). Note the relation to the aneurysm, close to, but separate from, the hilus of the kidney.

FIG. 3. (*E*) Photograph of the splenic artery aneurysm at the time of operation.

right kidney was not demonstrated (Fig. 5*C*). However, because of the characteristic pyelographic findings (Fig. 5, *A* and *B*), an exploratory operation of the right kidney was performed and disclosed a clotted calcific 1 cm. aneurysm of the right renal artery as it entered the hilus (Fig. 5*D*). Because preoperative studies had revealed decreased function in the left kidney and the left renal artery and kidney were hypoplastic, right nephrectomy was not done. A year following operation, the hypertension was still unrelieved and the patient was unimproved.

The second patient was a forty-eight

FIG. 4. *Arteriosclerotic hepatic aneurysm in a seventy-two year old man, visualized four years after its discovery at operation.* (*A*) Conventional frontal roentgenogram of the abdomen showing a rounded 1.5 cm. in diameter calcific area (arrow) between the eleventh and twelfth right ribs close to the border of the spine. (*B*) Intravenous abdominal aortogram showing tortuosity of the abdominal aorta and common iliac arteries. The hepatic artery is dilated at its origin, it then becomes aneurysmal in the calcific region (arrow). (*C*) Enlargement of the involved area showing the aneurysm (arrow). (*C* is reproduced with permission from *The New England J. Med.*[50])

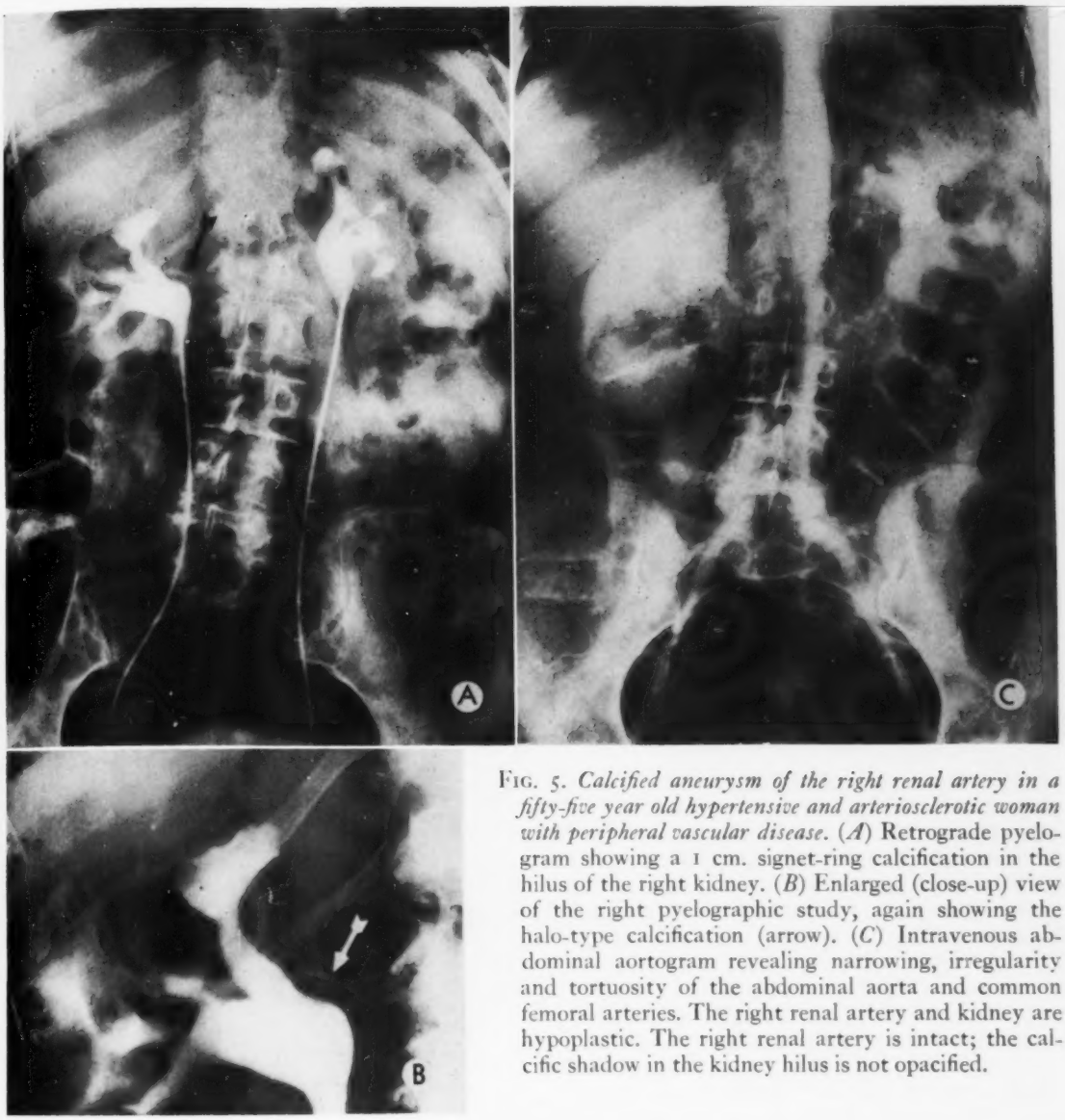

Fig. 5. *Calcified aneurysm of the right renal artery in a fifty-five year old hypertensive and arteriosclerotic woman with peripheral vascular disease.* (*A*) Retrograde pyelogram showing a 1 cm. signet-ring calcification in the hilus of the right kidney. (*B*) Enlarged (close-up) view of the right pyelographic study, again showing the halo-type calcification (arrow). (*C*) Intravenous abdominal aortogram revealing narrowing, irregularity and tortuosity of the abdominal aorta and common femoral arteries. The right renal artery and kidney are hypoplastic. The right renal artery is intact; the calcific shadow in the kidney hilus is not opacified.

year old man (Fig. 6, *A–C*), who also had hypertension (170/115 mm. Hg). Deformity of the right renal pelvis was demonstrated on a retrograde pyelogram (Fig. 6*A*). The intravenous abdominal aortogram revealed a 1 cm. aneurysm of the right renal artery (Fig. 6, *B* and *C*). This finding was confirmed at operation, but in this instance, because previous studies had demonstrated good left kidney function, a right nephrectomy was performed and the patient became normotensive.

Fig. 5.(*D*) Photograph at operation revealing the clotted calcified aneurysm (arrow).

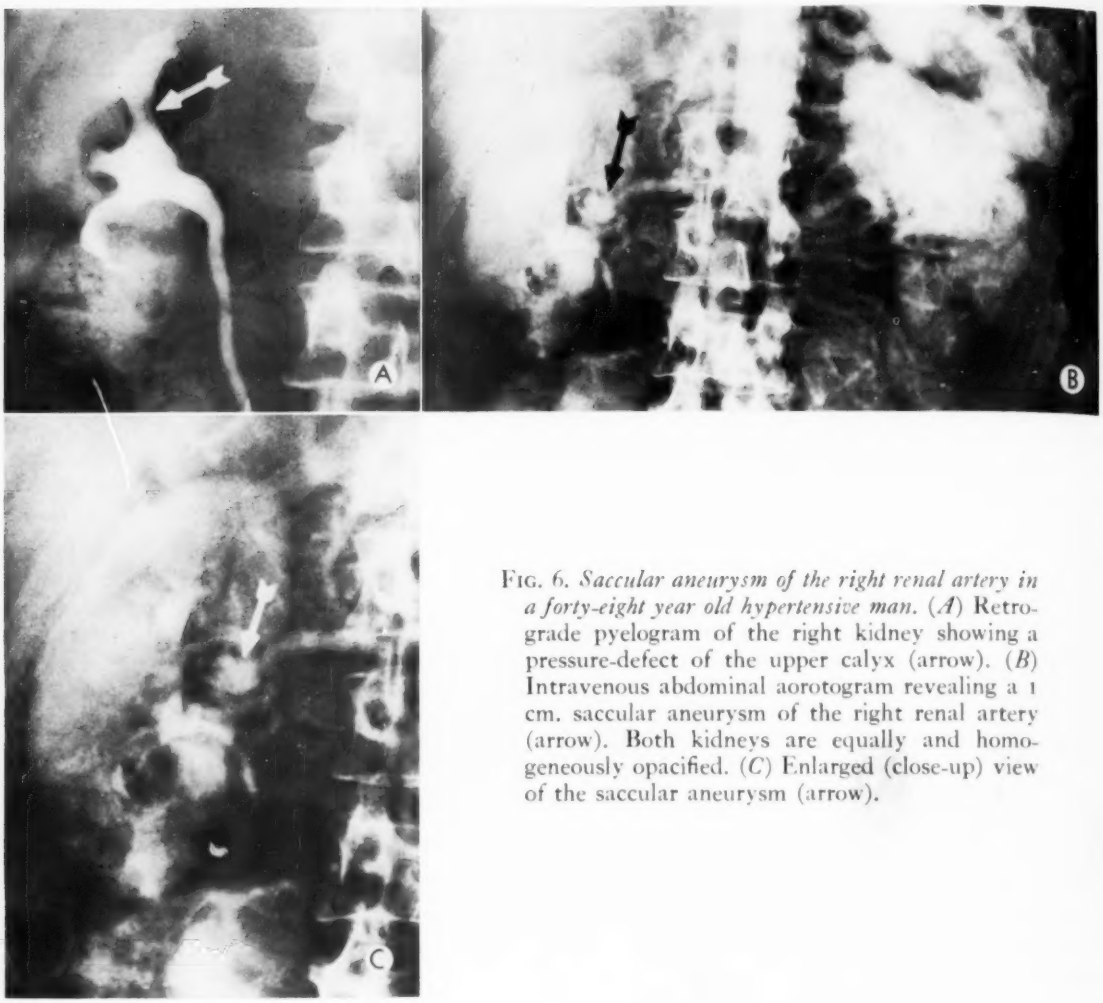

FIG. 6. *Saccular aneurysm of the right renal artery in a forty-eight year old hypertensive man.* (*A*) Retrograde pyelogram of the right kidney showing a pressure-defect of the upper calyx (arrow). (*B*) Intravenous abdominal aorotogram revealing a 1 cm. saccular aneurysm of the right renal artery (arrow). Both kidneys are equally and homogeneously opacified. (*C*) Enlarged (close-up) view of the saccular aneurysm (arrow).

DISCUSSION

Owens and Coffey,[37] in a thorough review of splenic artery aneurysms, discussed the incidence, pathogenesis, pathologic and clinical features and the tendency of these aneurysms to rupture during pregnancy. More recently, Culver and Pirson,[6] and Sheps and his co-workers[44] have reviewed the subject. There appears to be general agreement among surgeons that, once the diagnosis of splenic artery aneurysm is suspected, surgical exploration and excision of the aneurysm is essential to prevent rupture. Indeed, following the pioneer paper of Lindboe,[27] who emphasized that rounded, egg-shell calcific shadows in the left upper quadrant were likely to be aneurysms of the splenic artery, routine exploration of the abdomen and excision of the aneurysm and spleen have been advocated. Von Ronnen,[53] however, believes that only if the calcified splenic aneurysm is large, causes symptoms, or enlarges is surgery indicated.

Diagnosis of splenic artery aneurysms is dependent mainly upon roentgenography. In 15 per cent of the cases reviewed by Owens and Coffey,[37] there were valuable diagnostic roentgen clues consisting of either a pulsatile mass in the posterior wall of the stomach or round or oval-shaped thin lined (egg-shell) calcifications located in the upper left quadrant of the abdomen (Fig. 1, *A* and *B*; 2*A*; and 3, *A* and *B*).

Differentiation between gastrointestinal and renal calcifications can readily be made by contrast roentgen studies. Pancreatic stones are more difficult to differentiate,[53] but now that a method for opacifying the aorta via the intravenous route exists, the definitive diagnosis need not be delayed.

Owens and Coffey[37] also emphasize that awareness of the possible existence of a splenic artery aneurysm is very important in establishing the diagnosis. Among the clinical findings cited by these authors are pain in the left upper quadrant, flatulence and splenomegaly. These are so nonspecific that they provide little aid in diagnosis. Other signs of greater significance, although rare, are a palpable and occasionally pulsatile tumor in the left hypochondrium. However, the "egg-shell" opacities in the left upper quadrant, unrelated to the renal or gastrointestinal systems, provide the most significant clues of splenic artery aneurysms.

Of interest, is the case showing opacification of the splenic artery aneurysm (Fig. 1*B*) and not of the adjacent calcification. At operation the latter turned out to be a calcified lymph node. Multiple aneurysms of the splenic artery have been reported in the literature, but these have been round, lined by calcific walls and have contained mottled densities, in marked contrast to the irregularly shaped and often homogeneously calcified structure of the calcified lymph node (Fig. 1*A*).

Hepatic artery aneurysms are rare. Browning, Clauss and MacFee[2] in a recent report of 2 cases stated that they found 121 cases in the medical literature. Their 2 cases, 2 other cases reported since then,[17,24] the case of a child,[23] and the one case reported above bring the total to date to only 127. Notable reviews of the subject[12,16,30,40,45,46,51] indicate that males with hepatic artery aneurysms predominate at a ratio of 3 to 1. The average age is thirty-eight years. There also appears to be general agreement that the most common causes of hepatic artery aneurysms are infection (60 per cent), arteriosclerosis (22 per cent), and trauma (10 per cent).[30] The aneurysms may be intrahepatic or extrahepatic, the latter being more common and varying from a small size (a true aneurysm) to a huge (false type) hematoma. Both tend to be adherent to surrounding tissues by firm fibrous adhesions that compress the surrounding structures, especially the bile ducts. The majority of hepatic artery aneurysms are solitary; only in 11 instances were multiple aneurysms found. The hepatic artery itself rather than the branches is usually involved.

Hepatic artery aneurysms, like all aneurysms, have a tendency to rupture. The peritoneal cavity is a favorite site, and sudden shock and death due to hemorrhage are common. Direct perforation into the duodenum and stomach has also been reported.[9,15,52] Perforation may also occur into the biliary duct system and cause hematemesis or melena, or both.[31] As a result, a triad of symptoms referable to hepatic artery aneurysms has become recognized. These are right hypochondriac pain, gastrointestinal hemorrhage, and jaundice due to pressure on biliary ducts. However, these too, are so nonspecific that they are of little value in the differential diagnosis. A pulsating right upper quadrant mass with a systolic bruit and enlargement of the liver are more valuable but rarer findings. About 36 cases of hepatic aneurysm have been diagnosed during life,[2,24,26] all, as in our case at exploratory laparotomy. Accordingly, a high index of suspicion coupled with a contrast visualization study of the abdominal arterial tree promises to be the most effective way of making the diagnosis.

In our case (Fig. 4*A*), a right upper quadrant calcification accurately localized the site of the arteriosclerotic hepatic aneurysm. The American[22] and French[21] literature contains references to this sign. However, since calcification usually occurs only in the arteriosclerotic hepatic artery aneurysms and since these comprise only about one quarter of all aneurysms, it probably is an uncommon finding. Bruwer and

Hallenbeck,[4] after retrospective study of a case, suggested that a filling defect in the duodenum may also be of diagnostic significance. In our patient, however, probably because of the small size of the aneurysm, contrast duodenal studies were normal.

Recent accounts of survival following surgical correction of hepatic aneurysms indicate that a new era in treatment of this lesion has evolved.[2,11,13,18,24,26,38] Indeed, excision of the celiac axis for repair of an hepatic artery aneurysm has been reported.[18,24] Similarly, excision of an arteriosclerotic celiac artery aneurysm with anastomosis of the hepatic artery to the origin of the celiac axis was successfully performed by Schumacker and Siderys.[41] Accordingly, preoperative diagnosis is no longer of academic interest. In this connection, however, it must be mentioned that our patient with the arteriosclerotic aneurysm (Fig. 4, *A–C*) had been asymptomatic during the four year period following its discovery at laparotomy.

Although rare, aneurysms of the renal artery are being encountered more frequently. This is apparently due to the increasing use of aortography, especially in young hypertensives in order to determine if there is a renal vascular cause for the hypertension.[35,36] Harrow and Sloane[19] in 1959 reported 5 new cases, reviewed the literature and pointed out that because of the use of aortography over one-third of the total of 180 cases of renal artery aneurysms had been reported within the six year period from 1951 to 1957.

Ever since Renck's[39] classic description of the roentgen appearance of calcified renal artery aneurysms, a signet-ring, halo-shaped calcification just medial to the renal pelvis has become established as pathognomonic of the lesion (Fig. 5, *A* and *B*).[29,34,53] This sign constitutes strong evidence for renal artery aneurysms,[34] but only about 25 per cent of the cases of renal artery aneurysms show this calcification. Harrow and Sloane[19] recently found that 69 of the total 180 cases reported in the literature had been diagnosed because of the characteristic calcific ring shadow. Aortography, chiefly translumbar, established the diagnosis of 28 of the 180 cases.

In the absence of calcification, a renal artery aneurysm cannot be excluded and should be suspected when there is an unexplained deformity of a calyx (Fig. 6*A*). Contrast aortography of the abdominal aorta either by the intravenous, retrograde or translumbar routes is clearly indicated in such cases, especially because, in marked contrast to the calcified aneurysms of the renal artery, the noncalcified aneurysms have a tendency to rupture.[19] Accordingly, here too, a high incidence of suspicion that a renal artery aneurysm is the cause of an unknown hypertension, poor or nonfunctioning kidneys, unexplained extrarenal or intrarenal noncalcified space-occupying lesions is necessary.

DIFFERENTIAL DIAGNOSIS

In cooperative patients, intravenous abdominal aortography may be expected to provide a highly satisfactory contrast study of the abdominal aorta and its branches (Fig. 7, *A* and *B*). However, in a recent case of a twenty-six year old woman with hypertension, the tortuosity of the splenic artery prevented adequate visualization of the left renal artery (Fig. 8*A*). Accordingly, percutaneous transfemoral renal arteriography[17] was performed with the patient in a slightly oblique position, and stenosis of the proximal portion of the left renal artery with poststenotic dilatation of the distal segment of the left renal artery (Fig. 8*B*) was demonstrated. Splenectomy followed by anastomosis of the splenic artery to the poststenotic segment of the left renal artery resulted in the blood pressure returning to normal levels.

Tortuous linear calcifications of the splenic artery in the left upper quadrant due to arteriosclerosis are readily differentiated from aneurysms of the splenic artery (Fig. 9) by the regular pattern of the calcifications. An echinococcus cyst may appear exactly like a splenic artery

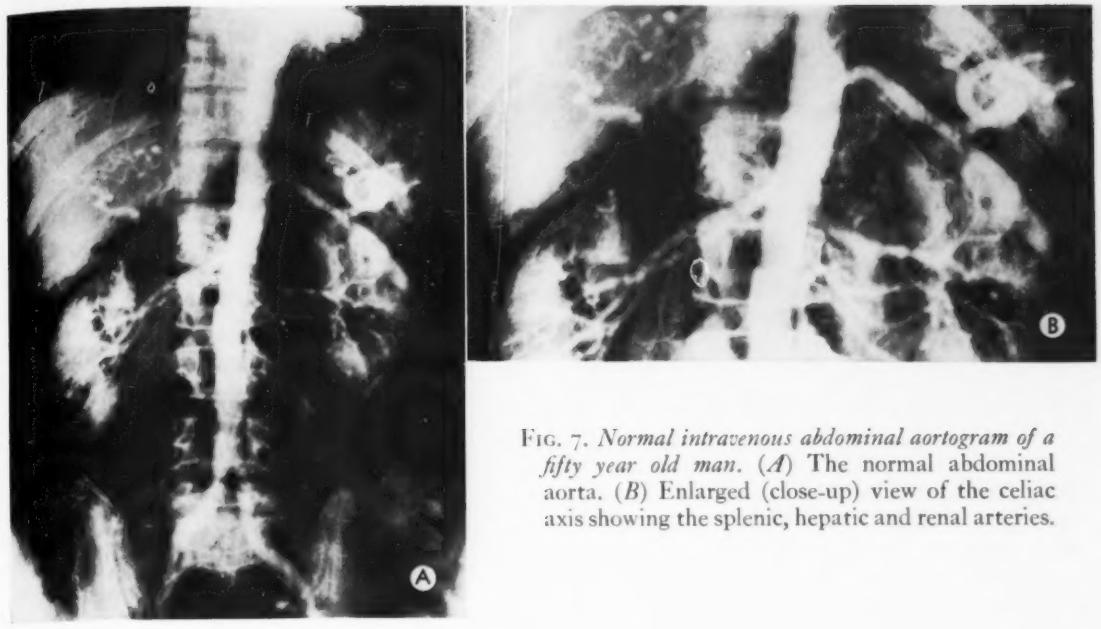

Fig. 7. *Normal intravenous abdominal aortogram of a fifty year old man.* (*A*) The normal abdominal aorta. (*B*) Enlarged (close-up) view of the celiac axis showing the splenic, hepatic and renal arteries.

aneurysm and be differentiated only when contrast materials fail to opacify it. In such an instance intravenous abdominal aortography (Fig. 10) by failing to opacify the egg-shell calcification excluded an aneurysm. Rarely, splenic artery aneurysms may be confused with renal artery aneurysms, especially if calcific shadows are in the vicinity of the left kidney and there are complaints referable to the urinary system.

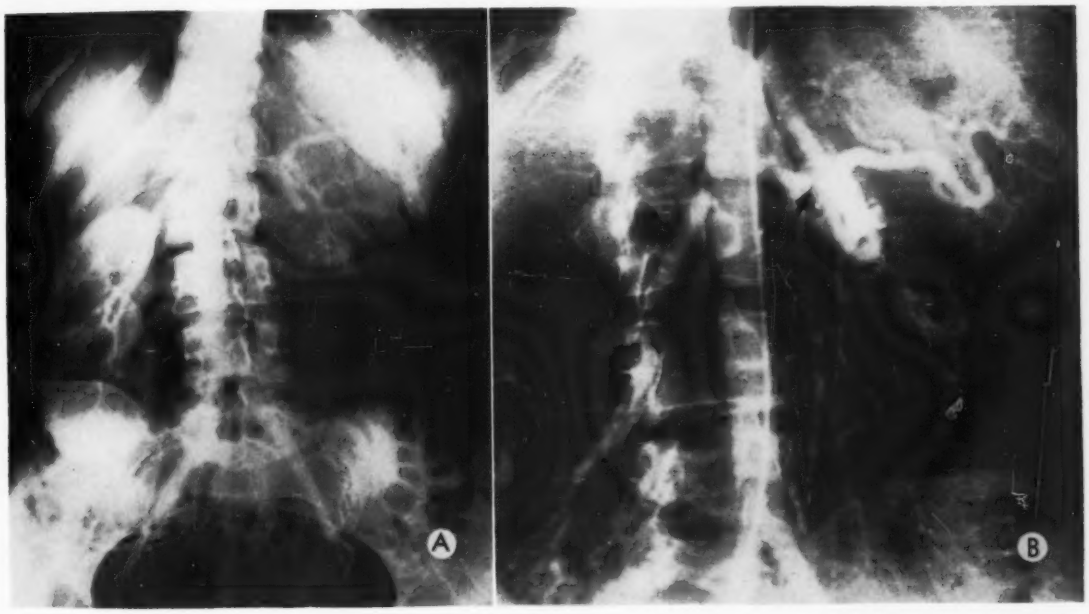

Fig. 8. *Hypertension due to left renal artery stenosis in a twenty-six year old woman.* (*A*) Intravenous abdominal aortogram showing tortuosity of the splenic artery obscuring the left renal artery (arrow). (*B*) Percutaneous translumbar renal arteriogram disclosing the site of stenosis of the left renal artery (arrow) with poststenotic dilatation of the distal portion of the renal artery.

FIG. 9. *Calcification and tortuosity of the splenic artery in a ninety year old physician.* The regular, though dilated, caliber of the artery is characteristic of arteriosclerotic involvement of the splenic artery and should be differentiated from saccular aneurysms of the splenic artery.

Usually, the larger size of the calcified lesion in the left upper quadrant (Fig. 3*A*) is against it being due to a renal artery aneurysm. Here, too, intravenous aortography readily established the diagnosis of splenic artery aneurysm, and operation proved it.

Gallstones may be confused with hepatic and renal artery aneurysms. The characteristic appearance of a laminated gallstone (Fig. 11) should not be difficult to recognize. Intravenous abdominal aortography in this patient was performed because of hypertension.

Admittedly, intravenous abdominal aortography does not achieve the high degree of radiopacity produced by translumbar or retrograde aortography. However, the morbidity of the translumbar approach is probably greater than that of intravenous abdominal aortography in certain patients. For example, one patient (Fig. 12) was found to have a right renal artery aneurysm in the course of retrograde aortography, performed in order to locate the site of coarctation of the abdominal aorta. Demon-

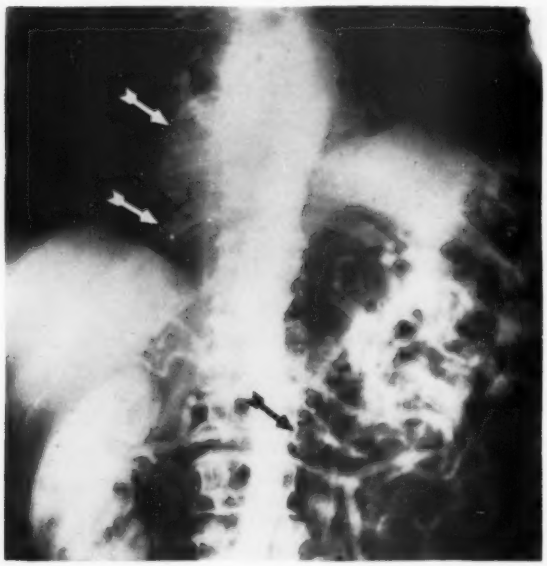

FIG. 10. *Egg-shell calcification in the left upper quadrant of the abdomen due to an echinococcus cyst (lower arrow) simulating a splenic artery aneurysm.* Note the opacified tortuous arteriosclerotic abdominal aorta. The splenic artery is also markedly tortuous, but the egg-shell calcification is not opacified. Note also (upper arrows) the mediastinal (nonopacified) calcification due to a huge mediastinal echinococcus cyst.

FIG. 11. *Intravenous abdominal aortogram revealing a tortuous abdominal aorta.* The left renal artery is hypoplastic and the left kidney is not opacified. A laminated gallstone in the right upper quadrant is easily differentiated from a right renal and/or hepatic artery aneurysm.

stration of the site of coarctation was successful, but a severe hypotensive reaction with renal shut-down following injection of diodrast was almost fatal.[14] Indeed, 2 fatalities in patients with coarctation of the abdominal aorta and renal artery stenosis after retrograde catheterization have recently been reported.[3] Accordingly, it would appear that intravenous abdominal aortography in such cases may be sufficient in establishing the diagnosis of coarctation of the abdominal aorta and renal artery stenosis without resorting to arterial catheterization.

SUMMARY AND CONCLUSIONS

A significant advance in the diagnosis and differential diagnosis of aneurysms of the splenic, hepatic and renal arteries has been achieved with intravenous abdominal aortography. This consists of rapid, simultaneous and bilateral injection of concentrated contrast organic iodides and precisely timed roentgenography. Inexpensive special needle-stopcock units, syringes and the use of apparatus ordinarily on hand for roentgen examination of the gastrointestinal or genitourinary systems make the procedure generally available and practical.

FIG. 12. *Retrograde abdominal aortogram revealing a saccular right renal artery aneurysm in a twelve year old girl with coarctation of the abdominal aorta.* (Reproduced with permission from *Surg., Gynec. & Obst.*[14].)

In a series of 350 consecutive patients who had had intravenous abdominal aortography, 3 were found to have arteriosclerotic aneurysms of the splenic artery; one had an arteriosclerotic hepatic artery aneurysm. Two hypertensive patients had renal artery aneurysms. All the cases of splenic artery aneurysm exhibited the classic roentgen features of arteriosclerotic artery aneurysms; namely, large, oval shaped, eggshell calcifications in the left upper quadrant. In one patient left upper quadrant calcifications were found during an incidental roentgen study of the abdomen. The second patient had vague epigastric discomfort. In the third, hematuria and the inability to dissociate the calcifications from the renal shadow made differential diagnosis more difficult. However, intravenous abdominal aortography was definitive. Excision of the splenic artery and the aneurysm, and splenectomy were curative in every case.

An arteriosclerotic hepatic artery aneurysm, found in one case during exploratory operation, was definitively diagnosed four years later by intravenous abdominal aortography. Excision of the aneurysm was not advised because of the asymptomatic state of the patient and because of advanced arteriosclerotic changes in the rest of the abdominal aorta.

A signet-ring calcification in the hilus of the right kidney in one case was strong evidence for the diagnosis of a renal artery aneurysm even though intravenous abdominal aortography failed to opacify the lesion. The diagnosis was confirmed at operation, but nephrectomy was not done because of severe abdominal aortic endarteritis with left renal artery and kidney disease. In this case, the relentless progress of the hypertension was not altered. In contrast, another patient with a saccular

aneurysm of the right renal artery, revealed by intravenous abdominal aortography, had excellent kidney function and a normal renal arterial blood supply in the opposite kidney. Nephrectomy resulted in the patient becoming normotensive.

Intravenous abdominal aortography is also important in the differential diagnosis of aneurysms from other lesions of the abdominal aorta and its branches. Failure to opacify calcified or egg-shell lesions which mimic splenic or renal artery aneurysms is good evidence against the diagnosis of aneurysm. Occasionally, a thrombosed aneurysm will fail to opacify. However, its proximity and relationship to the opacified vessel will allow proper diagnosis. Gallbladder, intrahepatic and renal stones may often be readily identified on contrast studies of the gallbladder and genitourinary system; calcified echinococcus cysts, mesenteric lymph nodes and pancreatic stones may prove more difficult to differentiate from the calcifications of aneurysms. Failure to opacify such lesions by aortography, of course, excludes aneurysms. Although aortography may also fail to opacify calcified and thrombosed renal artery aneurysms, fortunately, in such instances, the characteristic signet-ring (halo) calcification, located in the hilus of the kidney, is of great significance.

Finally, intravenous abdominal arteriography cannot always be relied upon to exclude renal arterial stenosis. These cases may probably be better evaluated by percutaneous transfemoral renal arteriography or translumbar arteriography.

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The authors are grateful to Drs. S. W. Moore and Victor F. Marshall who respectively gave permission for publication of the operative photographs (Fig. 3*E* and 5*D*).

ADDENDUM

Since this paper was submitted for publication, J. A. Spittel, J. F. Fairbairn, O. W. Kincaid and W. H. ReMine have reviewed the experience in 15 cases of splenic artery aneurysm at the Mayo Clinic (*J.A.M.A.*, 1961, *175*, 452–456). They, too, stress the importance of aortography in diagnosis.

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VOL. 86, NO. 6

EDEMA OF THE INTERURETERIC RIDGE—A USEFUL ROENTGEN SIGN*

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THE purpose of this paper is to call attention to edema of the interureteric ridge of the urinary bladder as a useful finding in roentgen diagnosis. The usual cause for the edema is a stone that has become impacted in the distal ureter or has recently passed through the ureteral orifice.

In 1948, Edling[1] reported his observations of the interureteric ridge. He described its normal appearance and the changes seen on 135 consecutive urograms of patients who had symptoms of ureteral calculi. He recorded the distinct bowed contour, continuous and cephalically concave, of the normal interureteric ridge. The distance between the ureteral contrast and the craniad contour normally was 2 to 3 mm., using a 15° caudocephalad projection of the roentgen-ray beam with the patient in the supine position.

In more than half of Edling's series the interureteric ridge was demonstrated. Swelling of the affected side of the interureteric ridge was present in 37 cases in which a ureterovesical calculus was seen. In 4 cases without visible stones the swelling was attributed to a recently passed or non-opaque calculus.

In 1954, Filho[2] discussed this sign and noted that swelling of the interureteric ridge subsided within forty-eight hours after passage of the ureterovesical calculus.

We have studied 125 unselected consecutive intravenous pyelograms in an attempt to determine the incidence of visualization of the interureteric ridge, the frequency of edema and the value of this sign in the diagnosis of ureteral calculus. The duration of the edema after the stone passed, the length of time required to develop the edema, and other factors, both anatomic and pathologic, that might simulate edema of the ridge were also studied.

MATERIAL AND METHODS

Following the intravenous injection of 30 cc. of 50 per cent hypaque, two pyelograms were made at ten minutes, one with and one without ureteral pressure, in the anteroposterior projection. A posteroanterior roentgenogram was made at fifteen minutes. A coned anteroposterior bladder roentgenogram was made without angulation at twenty minutes. All the roentgen studies were made at a 40 inch distance.

The sexes were equally distributed in series of 125 patients, and the ages ranged from two to eighty-seven years. The mean age of the 105 cases in which this information was recorded was forty-five years.

Measurements of the interureteric ridge were made from the bottom of the intramural portion of the ureter filled with contrast material to the nearest point of the roof of the interureteric ridge. The roof is seen as an opaque caudally convex structure above the lucent band of the ridge. When the ureter was not visible, the thickness of the band of lucency was measured. These points were often indistinct, so the measurements are not considered to be precise. From the point of view of establishing the normal measurements, however, this is not important because, in pathologic states, the deviations from normal were quite distinct.

RESULTS

The interureteric ridge could be seen in 45 of the 125 pyelograms (36 per cent). In the 36 normal cases in which it was measurable, it averaged 5 mm., with a normal

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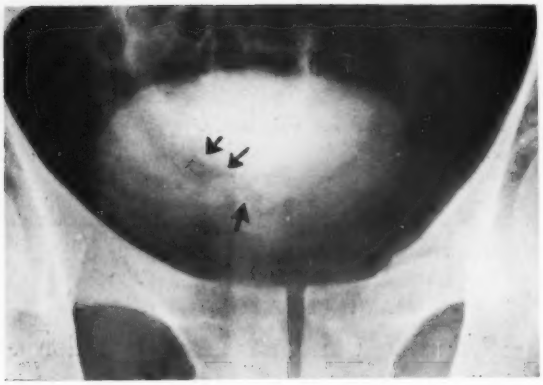

FIG. 1. H. M., a twenty-eight year old white male, had an acute onset of right flank pain and microscopic hematuria. The roentgenogram demonstrates calculus (lower arrow) and edema of the right interureteric ridge (upper arrows).

range of 2 to 9 mm. There were 12 patients with a ureterovesical stone or who had just previously passed a roentgenologically verified calculus. In 2 of these the ridge was not seen well enough to be evaluated; 8 showed edema of the ridge (Fig. 1, 2 and 3); and, in the remaining 2, in whom the studies were made five and seven days, respectively, after passage of the stones, there was no evidence of either calculus or edema. In all the cases with edema there was some retention of the contrast medium in the affected renal collecting system or complete absence of function on the affected side.

FIG. 2. J. G., a fifty-nine year old white male, had severe pain in the left lumbar region radiating to the left scrotum, and microscopic hematuria; a stone was passed spontaneously. The roentgenogram demonstrates edema of the interureteric ridge and partial ureteral obstruction.

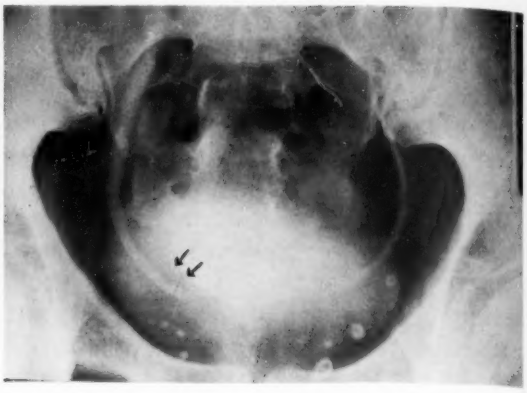

FIG. 3. P. C., a thirty-two year old white male, had severe pain in the right flank associated with frequency and microscopic hematuria; a stone was passed spontaneously. The roentgenogram indicates increased obliquity of the upper border of the interureteric ridge (arrows) associated with edema.

There was no difference in the sex distribution of those cases in which the ridge was visualized. This is contrary to Edling's results in which males predominated.

The bladder size, measured on the urograms, was graded from 1+ to 3+ in 83 cases. In 28 of these in which the ridge was visible, 19 were graded 1+ or 2+; of the remaining 55 in which the ridge was not visualized, 32 were so rated. This would appear to indicate that the ridge was visible relatively more often in those bladders that were not grossly distended. The impression was gained, confirming Edling's observations, that bladder distention was a significant factor in the failure of visualization of the interureteric ridge.

ROENTGEN ASPECTS OF THE URINARY BLADDER

The urinary bladder may present in the anteroposterior projection a number of different aspects. One may not see the interureteric ridge if the bladder is too distended or dilated. With the patient supine, stereoscopic and lateral cross-table roentgenograms show that the roof above the interureteric ridge represents the fundus of the bladder folded posteriorly over the relatively fixed trigone. The posteroanterior roentgenogram was of no assistance in the

visualization of the ridge in any instance. Oblique roentgenograms, in the few cases studied using this projection, showed the same findings as the anteroposterior roentgenograms. The opaque roof of the ridge may be confused with the edge of the overlying sacrum. The lucent interureteric ridge may be mimicked by an indentation of the inferior wall of the bladder which also casts a lucent shadow; however, it has a caudally concave lower border as compared to the convex shadow of the interureteric ridge and it always is lower in position (Fig. 4). Occasionally, the ridge may not be visualized on one examination and may be visible on another, but generally the findings are consistent on all examinations of the same patient.

When edema of the interureteric ridge is present, the affected side casts a radiolucent shadow ranging in size from 9 to 18 mm. Even more important than the width of the ridge for the recognition of edema is the alteration in the downward convexity on the affected side. Flattening and sometimes even concavity of the roof on the affected side may be quite striking. A unilateral radiolucent shadow in the region of the interureteric ridge may be caused by a ureterocele filling very slowly with contrast medium (Fig. 5). This type of defect is usually sharper in outline than edema of the ridge. It may not be accompanied by appreciable dilatation of the upper urinary tract.

FIG. 4. The radiolucent ridge (arrows) represents an indentation of the inferior wall of the bladder. This may be confused with the interureteric ridge when the lower ureters are not visualized.

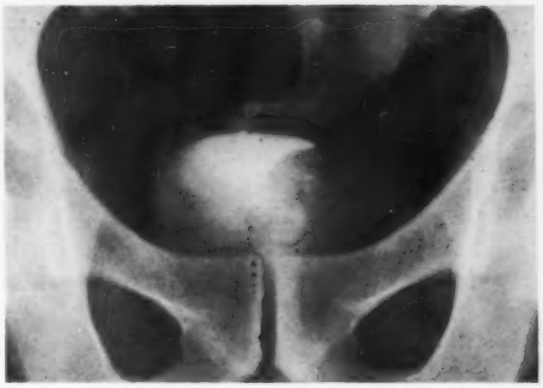

FIG. 5. J. H., a thirty-five year old white male, had pain in the left lower quadrant of the abdomen and microscopic hematuria. The roentgenogram demonstrates a ureterocele.

On occasion, extrinsic pressure from a distended intestine or from an adjacent mass lesion may cause widening of the ridge simulating the edema sign (Fig. 6). Carcinoma usually has sharper and often ragged edges.

DISCUSSION

The interureteric ridge was observed in 36 per cent of this series of 125 routine, unselected pyelograms. It was sometimes difficult to measure the ridge because the

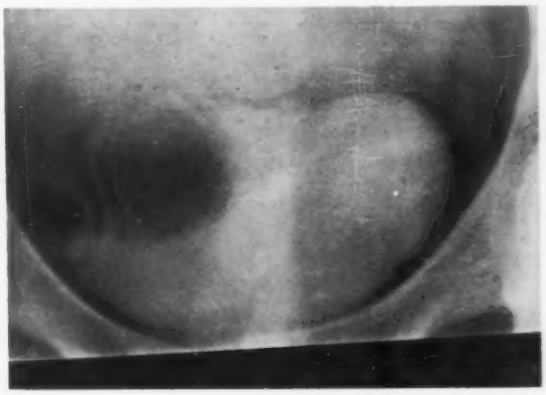

FIG. 6. E. M., a forty year old Negro female, had a palpable mass in the pelvis. The roentgenogram demonstrates extrinsic pressure on the bladder simulating edema or ureterocele. A fibroid tumor was removed and subsequent roentgen studies showed a normal appearing bladder.

roof could not be clearly seen or the distal end of the ureter was not filled with the medium. When the roof cannot be visualized the ridge cannot be measured or evaluated. In all the cases in which the ridge was visible and a calculus was present, the edema sign was evident.

The development of edema is probably rapid, since most of the roentgenograms demonstrating it were made shortly after symptoms began. However, we could not be certain how long the calculi were at the ureterovesical junction before the onset of edema. In 2 cases the edema subsided within a week; therefore, the edema sign would probably not be evident more than five days after passage of a stone.

The edema was always accompanied by other evidence of ureterovesical obstruction in the upper collecting system, such as hydronephrosis, a nephrogram or nonfunction. Hence, as an indication of ureterovesical calculus, it merely complements these other findings. The sign of edema of the ridge is most useful in the case where there is obstruction at the ureterovesical junction but where no calculus is seen. In these cases, the presence of edema would contraindicate a diagnosis of stricture, ureterocele, a fibrous band or carcinoma.

SUMMARY

Pyelograms of 125 consecutive cases were reviewed. An interureteric ridge was seen in 36 per cent. Edema of the interureteric ridge was observed in all cases of impacted or recently passed stones in which the ridge was visualized. This is a useful roentgen sign, particularly in the patient who has ureterovesical obstruction without a visible calculus.

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CHANGES IN HUMAN RED BLOOD CELLS DURING IN VITRO EXPOSURE TO SEVERAL ROENTGENOLOGIC CONTRAST MEDIA*

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THE dangerous reactions which are sometimes observed following intravascular administration of roentgenologic contrast media are poorly understood. In a recent review of the problem, Read[13] has pointed out that the mortality following cardioangiography and arteriography is very much higher than for roentgenographic procedures in which the contrast medium is administered into a peripheral vein. He reports animal experiments designed to study the direct effects that contrast media might have upon the blood itself and upon the vessels into which it flows before appreciable dilution has been effected by mixing within the circulation. Read places considerable emphasis upon the red blood cell "agglutination" that was observed microscopically in the small vascular channels close to the site of injection, and he suggests that this "agglutination" may be of major importance in the etiology of some of the serious clinical reactions.

The purpose of the present investigation has been to study *in vitro* some of the effects of various contrast media upon the human red blood cells. The report includes observations on the size, shape and agglutinability of red blood cells, penetration of contrast medium across the red blood cell membrane, and indications of red blood cell damage as evidenced by *in vitro* hemolysis and by impaired *in vivo* red blood cell survival.

METHODS

Contrast Media. Four contrast media* were studied:

Brand Name	*Composition*
urokon	sodium acetrizoate
miokon	sodium diprotrizoate
hypaque	sodium and N-methyl diatrizoate
ditriokon	sodium diatrizoate sodium diprotrizoate
"triple medium"	sodium acetrizoate sodium diprotrizoate sodium and N-methyl diatrizoate

The "triple medium" was compounded by the authors for comparative *in vitro* tests (see Fig. 4) and consisted of urokon, hypaque and miokon in final concentrations of 23.3 per cent, 25 per cent and 23.3 per cent, respectively.

Normal red blood cells were obtained from laboratory personnel known to be free from hematologic disease. Freshly drawn blood was either defibrinated with glass beads, or placed directly into acid citrate dextrose anticoagulant solution (ACD, U.S.P. Formula A). Unless otherwise stated, the blood was used within 24 hours of procurement.

Hemoglobin concentration of whole blood was determined as cyan-hemoglobin, according to Drabkin.[8] The hemoglobin concentrations in plasma or in saline wash solutions were determined by the method of Crosby and Furth[7] for levels greater than 4 mg. per 100 ml., and by the method of Hanks *et al.*[10] for levels below 4 mg. per 100 ml. It was previously determined that hemoglobin measurement by these methods was unaffected by the presence of contrast media in the solution to be tested.

Hematocrits on mixtures of red blood cells and contrast media were determined in

* Manufactured by the Mallinckrodt Chemical Works.

* From the Division of Hematology, Departments of Medicine and Preventive Medicine, Washington University School of Medicine, and the Mallinckrodt Institute of Radiology, St. Louis, Missouri.
Supported by Research Grant C-2918 (C2 and C3) from the National Cancer Institute of the Public Health Service.

microhematocrit tubes (75×1.5 mm.), centrifuged for 5 minutes at 12,000 r.p.m. (approx. 9,000 g. to the midpoint of the hematocrit tube) in an International Centrifuge, model 275. Hematocrits on blood samples obtained during blood volume measurement were determined in Wintrobe tubes centrifuged for 55 minutes at 2,200 r.p.m. in a centrifuge of radius 25 cm. to the bottom of the centrifuge cups.

Mean corpuscular hemoglobin concentration (*M.C.H.C.*) was calculated in the usual manner,

$$\text{M.C.H.C.} = \frac{\text{hemoglobin, gm. per 100 ml.} \times 100}{\text{vol. packed red cells, ml. per 100 ml.}},$$

based on duplicate determinations of hemoglobin concentration and of hematocrit. Elevation in M.C.H.C. following exposure to hypertonic solutions was attributed to loss of intra-erythrocyte water.

Osmolarity was determined employing a Fiske osmometer. All samples were measured after appropriate dilution to provide levels of approximately 300 m.osm./per kg. of H_2O.

Specific gravity of concentrated solutions of contrast media was determined by triplicate weighing of accurately measured 5 ml. volumes and comparison against distilled water weighed under identical conditions.

Quantitative Measurement of Intracellular Penetration of Contrast Medium. Six 12 ml. aliquots of fresh defibrinated blood were centrifuged for 1 hour at 2,200 r.p.m.; the supernatant plasma and upper red blood cell layers were aspirated so as to leave 5.0 ml. of packed red blood cells remaining in each tube. Under these conditions it is known[5] that trapped saline makes up < 2 per cent of the packed cell mass. Sufficient I^{131}-tagged urokon* was added to each of three urokon solutions (final urokon concentrations 2 per cent, 10 per cent, 20 per cent) to provide a convenient counting rate. To each of two tubes containing 5 ml. of packed red blood cells was added 5 ml. of a tagged urokon solution. The contents were thoroughly mixed; after room temperature incubation of one tube for 5 minutes, the other for 30 minutes, aliquots were removed for measurement of hemoglobin and hematocrit and the tubes were centrifuged for 40 minutes at 2,200 r.p.m. The penetration of urokon into the cells was determined from the decrease in radioactivity of the supernatant fluid obtained after centrifugation compared with the original urokon solution. Appropriate corrections for the shift of water out of the cells were made on the basis of M.C.H.C. values determined on the original red blood cells and on the aliquots removed after the 5 and 30 minute periods of incubation. Intracellular water was assumed to equal 65 per cent of the true original red blood cell mass. Radioactivity was measured in a well-type scintillation counter.

In vivo red cell survival was determined using Cr^{51}, essentially as described by Ebaugh and co-workers.[9] After exposure for 30 minutes to 50 μc of Cr^{51} as sodium chromate, tagged normal red blood cells were incubated with the contrast media and then washed three times with 20 volumes of saline. Ten milliliters of tagged red blood cell suspension were injected from a calibrated syringe. Expected immediate 100 per cent survival was estimated as:

$$\text{expected counts per ml. of recipient's blood} = \frac{\text{total counts injected}}{\text{blood volume (ml.)}}.$$

Plasma volume was measured by the Evans Blue dye method, and blood volume calculated employing the hematocrit corrected for plasma trapping[5] and for the body/venous hematocrit ratio.[6]

RESULTS

Gross Observations. When a volume of whole blood was mixed with an equal volume of any of the four concentrated (*e.g.*, 70 per cent) contrast media, there was no immediate aggregation of red blood cells visible grossly or microscopically. However, after the mixture had stood undisturbed for

* Supplied by Abbott Laboratories.

several minutes, aggregates of red blood cells were observed in the film of blood on the wall of the tube above the fluid level. If the mixture was allowed to stand undisturbed for periods up to 1 hour, the clumps of red blood cells increased in number and in size. Mixing of the contents by repeated inversion of the test tube caused rapid dispersion of the red blood cell aggregates. When the mixtures were centrifuged after varying intervals of incubation, the red blood cells migrated to the top of the fluid layer and adhered together in a "slime" which broke up into smaller aggregates following vigorous remixing (Fig. 1). Progressively severe hemolysis was observed in the underlying fluid following centrifugation, depending on the length of incubation of the mixtures. The spontaneous aggregation of red blood cells was independent of the presence of plasma and could be largely prevented by repeated slow inversion of the tube during the first 5 to 10 minutes following addition of the blood to the contrast medium. Spontaneous clumping of red blood cells did not occur during subsequent undisturbed incubation, but comparable hemolysis was observed after centrifugation of the mixtures, and the centrifuged red cells formed an overlying "slime," as previously. It therefore appeared that spontaneous aggregation of red blood cells was favored if they were left undisturbed immediately following contact with high concentrations of contrast medium, presumably during the period of acute changes in the cells' size and surface properties. Once the red blood cells achieved a relatively "steady state," the forces of mutual attraction were greatly diminished and were manifested only when the red blood cells were brought into close contact by centrifugal force. The phenomenon of red blood cell lysis was largely independent of the aggregation, since it was of comparable severity in mixtures in which clumping was prevented by gentle motion of the test tube.

Microscopic Observations. Immediately upon exposure to high concentrations of contrast medium, the red blood cells examined in a free drop of fluid lost their biconcave shape and appeared initially as flat irregular plates or small dense spheres. When examined in a thin layer under a glass cover slip, all of the cells appeared as small dense spheres. After a sufficient period of exposure, hemolysis of cells occurred as evidenced by rapid fading of the cell color, leaving a faintly visible shrunken ghost. When an aggregate of adherent red blood cells was examined in a free drop of contrast medium, large numbers of cells were seen congregated together in tangential contact, but without the close approximation of cell walls and without the cellular distortion characteristic of immune agglutination. There was no rouleau formation. The application of a glass cover slip caused complete dispersion of all but the largest aggregates, indicating the weakness of the forces responsible for the aggregation. When red blood cells were exposed to solutions of NaCl, $Na_3C_6H_5O_7$ or dextrose of osmolarities equivalent to the concentrated contrast media, entirely similar changes in cell size, shape and properties of adherence were observed, but without significant hemolysis, even when exposure was extended over a period of several hours.

Fig. 1. Red blood cell aggregation following exposure to concentrated contrast medium. The red blood cells in the upper tube were exposed to urokon, centrifuged lightly and resuspended by inversion of the tube. Note the irregular cellular aggregates; the gray background is in part due to hemolysis. Evenly dispersed control cells suspended in saline are shown in the lower tube; the clear background indicates absence of hemolysis.

Reversibility of Morphologic Changes. The

concentrated contrast media were removed by washing the red blood cells three times in 20 volumes of isotonic sodium chloride solution. Striking further hemolysis of cells occurred during the first two washes, diminishing with the third wash (see ahead for quantitation of hemolysis). When the washed cells were resuspended in their native plasma, most of them regained their original size and biconcave shape and formed rouleau normally. However, the longer the period of exposure to the concentrated contrast media, the greater the number of cells which remained as small dense refractile spheres, suggesting irreversible damage.

Agglutinability of Cells. On several occasions, red blood cells from a group A_1 donor were exposed for various periods to concentrated contrast media, then washed three times, and the recovered cells made up to an accurate 2 per cent suspension in saline and compared with a 2 per cent suspension of unexposed cells against multiple dilutions of an anti-A serum. There was no evidence of impaired agglutinability of the recovered cells (a representative result is shown in Table I).

Quantitative Evidence for Cell Shrinkage. Five milliliter volumes of packed washed red blood cells were mixed with equal volumes of 14 per cent, 20 per cent and 26 per cent solutions of urokon. Samples were removed for duplicate hemoglobin and microhematocrit determinations after 5, 15 and 30 minutes of incubation. Progressive shrinkage of the red blood cells was observed as indicated by the rises in mean corpuscular hemoglobin concentration (M.C.H.C.) shown in Table II. Attempts to extend these observations to higher concentrations of the contrast medium were frustrated by the high density of the medium, which prohibited satisfactory sedimentation of the red blood cells by centrifugation. However, Figure 2 indicates that similar changes occurred in red blood cells exposed to somewhat higher concentrations of contrast media. Initially the red blood cells (specific gravity approximately 1.095) floated on top of the media. However, during incubation, loss of water from the red blood cells due to osmotic shrinkage resulted in increased cellular specific gravity and decreased specific gravity in the suspending medium, which favored an increasing number of cells being driven to the bottom of the hematocrit tube by centrifugal force. The calculated changes in intracellular and extracellular specific gravities occurring in response to the shift

TABLE II

RED BLOOD CELL SHRINKAGE DURING EXPOSURE TO CONTRAST MEDIUM

Concentration of Urokon	M.C.H.C.* (per cent) after incubation			
	Control	5 min.	15 min.	30 min.
14%	32.5	33.5	33.9	35.4
20%	32.5	34.2	35.3	40.5
26%	32.5	35.8	40.0	42.6

* Mean corpuscular hemoglobin concentration.

TABLE I

AGGLUTINABILITY OF RED BLOOD CELLS AFTER EXPOSURE TO CONTRAST MEDIUM

	Dilutions of Anti-A Serum							
	1/1	1/2	1/4	1/8	1/16	1/32	1/64	1/128
A_1 control cells	4	4	4	3–4	3	2	1	±
A_1 cells recovered after exposure to 40% urokon for 40 minutes	4	4	4	3–4	3	2–3	1	1

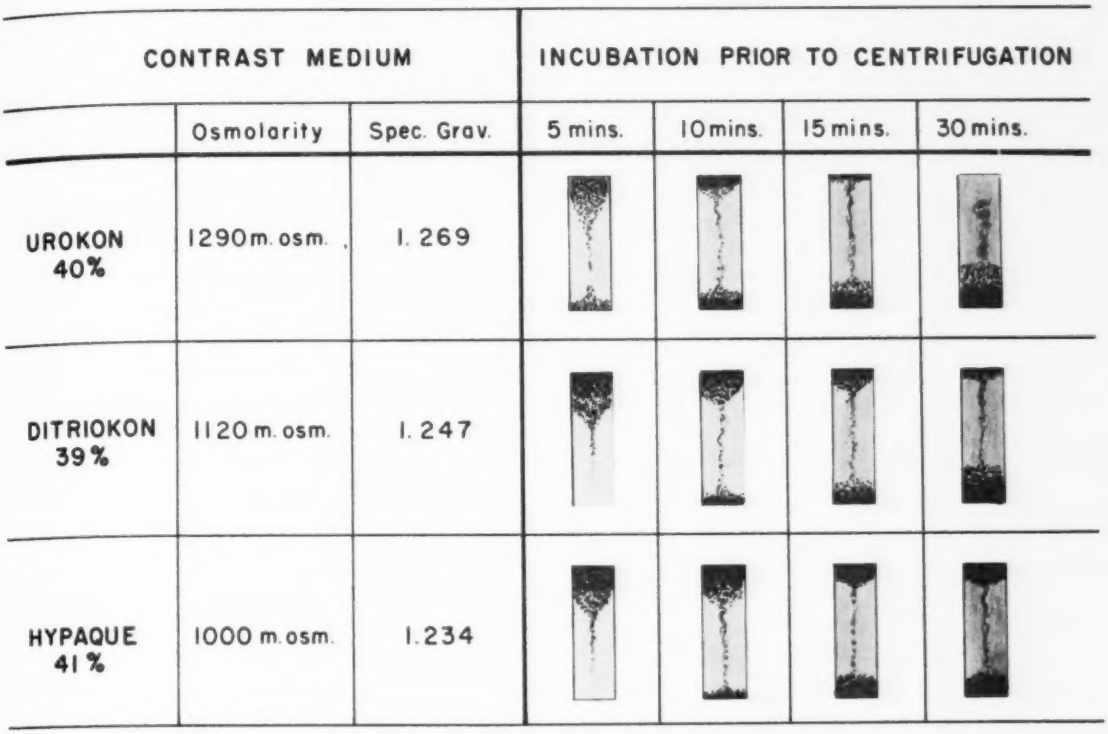

FIG. 2. Sedimentation of red blood cells by centrifugation after increasing periods of exposure in three contrast media. Blocks are diagrammatic representations of hematocrit tubes following centrifugation at 9,000 g. Note that following short periods of incubation the centrifuged red blood cells remain near the top of the tube; as the incubation period is prolonged, increasing numbers of cells can be centrifuged to the bottom of tube. The gray background represents increasing hemolysis with longer periods of incubation.

of water out of the cells were insufficient to account for the successful sedimentation of red blood cells in the relatively high concentrations of contrast media. It therefore seemed likely that some of the relatively heavy contrast medium had penetrated inside the cells.

Evidence that Urokon Penetrates Red Blood Cells. Fresh red blood cells were incubated for 45 minutes with equi-osmolar solutions of urokon, sodium chloride (a penetrating salt), trisodium citrate (a non-penetrating salt) and dextrose (which penetrates the red blood cell membrane, but does not come to complete equilibrium at high dextrose concentrations). The results, summarized in Table III, illustrate that the highest M.C.H.C. (44.6 per cent), indicating the greatest red blood cell shrinkage, was observed in the solution containing the citrate ion, to which the red blood cell membrane is impermeable. By contrast, the shrinkage of red blood cells in the solution containing urokon resembled that observed in the solutions containing the penetrating dextrose molecule and chloride ion.

Quantitative measurement of penetra-

TABLE III

SHRINKAGE OF RED BLOOD CELLS FOLLOWING 45 MINUTES INCUBATION IN FOUR HYPERTONIC SOLUTIONS

	Osmolarity (m.osm./kg.H_2O)	M.C.H.C.* (per cent)
20% urokon	630	35.6
1.84% NaCl	630	37.7
10.8% dextrose	630	34.9
6.56% $Na_3C_6H_5O_7 \cdot 5\frac{1}{2}H_2O$	630	44.6

* Mean corpuscular hemoglobin concentration.

TABLE IV

PENETRATION OF UROKON ACROSS THE RED BLOOD CELL MEMBRANE

	Incubation (min.)	Intracellular Urokon Concentration (I.U.C.) (per cent)	Extracellular Urokon Concentration (E.U.C.) (per cent)	Ratio I.U.C. /E.U.C.
2% urokon	5	0.25	1.8	0.139
	30	0.28	1.79	0.156
10% urokon	5	2.62	7.9	0.332
	30	2.75	7.68	0.358
20% urokon	5	6.0	14.9	0.402
	30	6.6	14.3	0.462

tion was accomplished employing I^{131}-tagged urokon, as described in detail under Methods. Observations were made after 5 and 30 minutes' incubation of red blood cells in final urokon concentrations of 2 per cent, 10 per cent and 20 per cent. The results are summarized in Table IV, where it is evident that the contrast medium penetrated into the red blood cells and that most of the penetration occurred within the first 5 minutes of exposure. The 0.139 and 0.156 ratios of intracellular and extracellular urokon concentration at an initial urokon concentration of 2 per cent indicate the maintenance of a steep gradient across the cell membrane; the higher ratios (0.332 and 0.402) obtained with the 10 per cent and 20 per cent concentrations of urokon may be indicative of progressive red blood cell damage. No attempt was made to extend these studies to urokon concentrations higher than 20 per cent because of the difficulty of accurate determination of M.C.H.C. at the high specific gravities of the more concentrated urokon solutions, and because gross hemolysis indicated profound cellular damage. The red blood cells exposed to 10 per cent tagged urokon were subsequently washed three times with untagged 10 per cent urokon. Considerable exchange of the intracellular tagged urokon occurred during the washing procedure, but at the conclusion of the third wash it was determined that the intracellular concentration of urokon I^{131} was three times greater than in the supernatant wash solution.

Quantitation of Hemolysis. When red blood cells were exposed to high concentrations of contrast media, increasing hemolysis occurred with increasingly long periods of exposure. When the red blood cells were replaced in a physiologic medium (*e.g.*, plasma, or 0.85 per cent NaCl), further hemolysis occurred. To illustrate the extent of the hemolysis, packed washed red blood cells were incubated with an equal volume of contrast medium for 40 minutes, then washed three times with 20 volumes of 0.85 per cent NaCl solution. The total free hemoglobin in each wash solution was measured and expressed as a percentage of the total hemoglobin present in the original intact red blood cell mass. In Figure 3, three contrast media adjusted to the same osmolarity (1830 m.osm./kg. H_2O) are compared with equi-osmolar

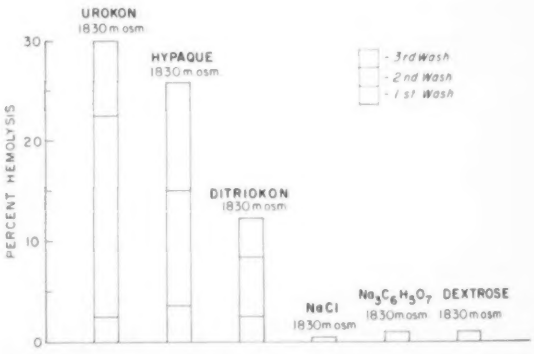

FIG. 3. Hemolysis following 40 minutes exposure to equi-osmolar solutions. The hemoglobin present in each wash solution is expressed as a per cent of the total hemoglobin present in the original red blood cell mass. There was so little hemolysis following exposure to equi-osmolar solutions of NaCl, $Na_3C_6H_5O_7$ and dextrose that the results of three washes were combined in drawing the three blocks on the right.

solutions of NaCl, $Na_3C_6H_5O_7$ and dextrose. Evidently, there are substantial differences between contrast media as to their ability to damage red blood cells as manifested by hemolysis (compare urokon and ditriokon). Furthermore, the hemolytic effect is much in excess of what could be attributed to their hypertonicity; for minimal hemolysis occurred when red blood cells were exposed to equi-osmolar solutions of NaCl, $Na_3C_6H_5O_7$ and dextrose. In Figure 4, similar observations are recorded for urokon, ditriokon and hypaque in concentrations supplied for use in clinical angiography. In addition, there are shown the effects of 70 per cent miokon (usually supplied as a 50 per cent solution because of its relative insolubility). On the extreme right is shown a "triple medium" composed of urokon, hypaque and miokon in final concentrations of 23.3 per cent, 25 per cent and 23.3 per cent, respectively. When the latter medium and ditriokon (which is a combination of hypaque 36.7 per cent and miokon 31.4 per cent) are compared with urokon, it is apparent that hemolysis may be diminished in the presence of combinations of organic iodide compounds, where no single compound is present in very high concentration. Finally, Figure 5 illustrates the extent to which hemolysis is dependent on the concentration of the contrast medium. Less than 5 per cent hemolysis occurred after 40 minutes of exposure to 33 per cent urokon, but hemolysis exceeded 80 per cent after comparable exposure to 50 per cent urokon.

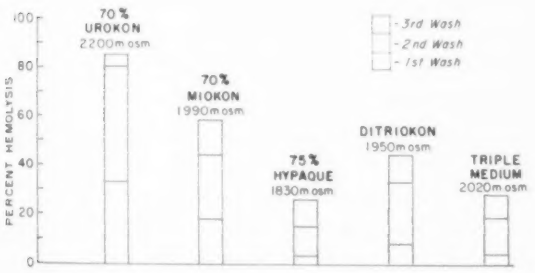

FIG. 4. Hemolysis following 40 minutes exposure to concentrated contrast media. The relatively small amount of hemolysis observed with the "triple medium" (which contains 23.3 per cent urokon, 23.3 per cent miokon and 25 per cent hypaque) may indicate a less damaging effect of combined contrast media, in which no single medium is present in high concentration.

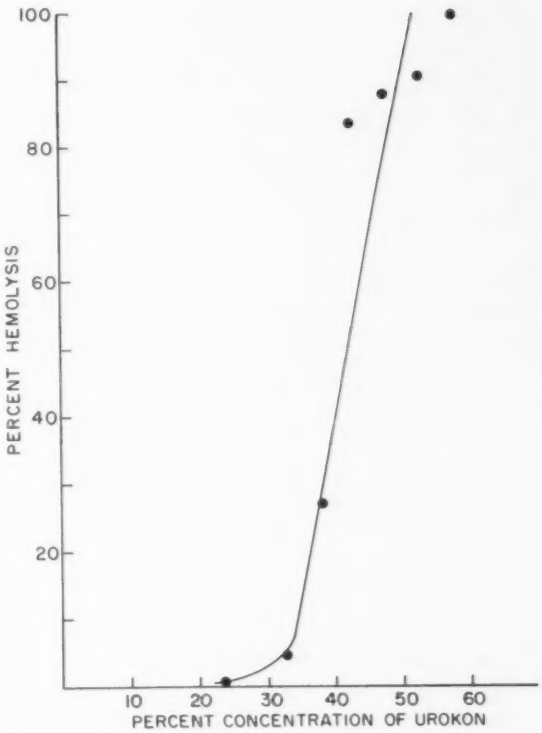

FIG. 5. Relation of hemolysis to concentration of urokon. Points on the curve represent the combined hemolysis occurring in three washes following 40 minutes exposure to urokon.

The extent to which hemolysis occurs following the clinical use of concentrated contrast media must be very small. On four occasions, samples of venous blood were collected within 20 minutes of completion of clinical angiographic studies, and in no instance did the plasma hemoglobin concentration exceed 0.7 mg. per 100 ml. These findings indicate negligible intravascular erythrocyte destruction (*i.e.*, certainly less than 0.25 ml. of red blood cells).[4]

Effect of Urokon on Red Blood Cell Viability. Normal red blood cells tagged with Cr^{51} were exposed to 45 per cent urokon for 1 minute on one occasion and for 45 minutes on another occasion; thereafter they

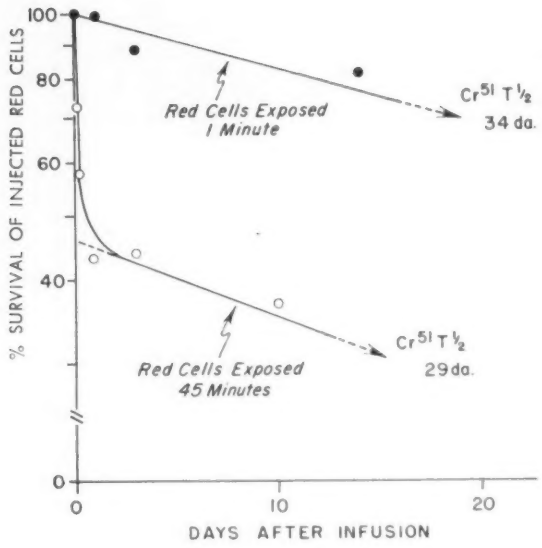

FIG. 6. *In vivo* survival of red blood cells recovered after exposure to 45 per cent urokon. During exposure and three subsequent saline washes prior to undertaking the *in vivo* survival studies, there was approximately 10 per cent hemolysis of the cells exposed to urokon for 1 minute and 50 per cent hemolysis of cells exposed to urokon for 45 minutes.

were washed 3 times with saline, resuspended in plasma and reinjected into the original donor (see Methods). The cells exposed to urokon for only 1 minute underwent approximately 10 per cent hemolysis prior to injection (*i.e.*, during exposure and washing), as compared to approximately 50 per cent hemolysis of the cells exposed for 45 minutes. The *in vivo* survivals of the injected cells are shown in Figure 6. Evidently there was no serious damage to the cells which were not hemolyzed during 1 minute's exposure to urokon and the subsequent washing procedure; they exhibited the expected 100 per cent immediate survival, and the Cr^{51} $T\frac{1}{2}$ followed over the succeeding 13 days was entirely normal (34 days). However, of the injected cells recovered after exposure to urokon for 45 minutes only 73 per cent were present in the circulation 10 minutes after injection, 58 per cent three hours after injection and 43 per cent 21 hours after injection. Not all of the cells were irreversibly damaged, for those which were present at 21 hours went on to survive normally as observed over the ensuing 12 days (Cr^{51} $T\frac{1}{2}$ 29 days).

Observation of the Rabbit Nictitating Membrane after Intracarotid Injection of Urokon. Injections were made into the cannulated carotid artery of the nembutalized rabbit, with continuous observation of arterioles, capillaries and venules employing a Zeiss slit lamp with green filter at 16× magnification. There is normally an irregular pulsatile pattern in the membrane vessels, and some segmentation of the red blood cell column is normally observed when the flow through a particular vessel complex is temporarily slowed. Sterile pyrogen free physiologic saline was injected rapidly in amounts of 0.2, 0.4, 0.8 and 1.0 ml. The only effect, observed mainly following the larger injections, was moderate temporary (30–60 seconds) spasm of the arterioles, with consequent slowing of flow in the capillaries and collecting venules and with accompanying segmentation of the red blood cell column during the intervals of retarded flow. Injection of 70 per cent urokon, even in amounts of 0.2 and 0.4 ml., was generally accompanied by an irritative reaction consisting of transient generalized muscular contractions, associated with more severe and prolonged spasm of the membrane arterioles. When 0.8 and 1.0 ml. of 70 per cent urokon were injected, the rabbit often experienced a generalized convulsion. Again, during periods of retarded blood flow secondary to arteriolar spasm, segmentation of the red blood cell column in the capillaries and venules was observed. However, as soon as the arterioles resumed their normal diameter (after 1 to 3 minutes), normal blood flow was promptly reestablished. The observers saw no evidence of sludging, or "agglutination" of the red blood cells that could be definitely attributed to a direct effect of the urokon causing *in vivo* intravascular aggregation of the cells.

DISCUSSION

The physiologic mechanisms underlying untoward clinical reactions to roentgenologic contrast media remain somewhat obscure. While some of the reactions (*e.g.*, urticarial) are almost certainly allergic in origin, others (*e.g.*, hemiplegia) appear to result from damage to areas in which the vascular supply is in the direct route of the injected material. The present investigation throws no light on the problems of allergic reactions; its purpose has been to explore possible ways in which the contrast media might cause direct damage to intact viable cells.

The human erythrocyte can be considered a relatively "tough" cell by comparison to most other soft tissue cells. It withstands refrigerated storage outside the human host for 3–4 weeks in relatively simple preservative solutions without great loss of viability. It is able to withstand striking changes in cell volume due to gain or loss of water, a property related in part to its existence as a "free cell" without intimate attachment to adjacent cells, and in part to its unique spatial form as a biconcave disk. It is interesting therefore to consider the changes in size, shape, membrane characteristics, and viability brought about after exposure to contrast media in current clinical usage.

Fresh erythrocytes were employed throughout the present investigation, for it was observed that stored red blood cells exhibited increased susceptibility to cellular aggregation and more rapid and extensive hemolysis than fresh red blood cells tested under identical conditions. The effects of the concentrated contrast media were manifested rapidly, as evidenced by the acute shrinkage of the red blood cells (observed microscopically to occur within seconds), the relatively prompt penetration of the organic iodide compound across the red blood cell membrane (Table IV), and the hemolysis (up to 10 per cent) that occurred when exposure was allowed for only 1 minute and then terminated by rapid dilution and repeated washing in physiologic saline solution. Undoubtedly some of the hemolysis observed during the washing procedures reflected the "paradoxical hypotonic hemolysis" discussed by Soderstrom,[15] and encountered so dramatically during the washing of red blood cells which had been suspended in high concentrations of penetrating polyhydric alcohols.[3,11] When intracellular osmolarity has been elevated by suspending cells in hypertonic solutions of a substance capable of penetrating across the cell membrane, sudden re-exposure of the cells to "physiologic" osmolarity may result in extremely rapid entry of water into the cells so that they swell and burst. This type of hemolysis can be largely eliminated by very slow addition of the physiologic wash solution, thereby permitting a gradual lowering in the osmolarity of the suspending fluid and allowing establishment of equilibrium between intracellular and extracellular solute. However, when physiologic saline was added very slowly to red blood cells which had been suspended in concentrated contrast media, only a modest decrease in hemolysis was achieved, suggesting that osmotic effects were not primarily responsible for the observed hemolysis. The failure of comparably hypertonic sodium chloride, sodium citrate and dextrose solutions to provoke significant hemolysis (Fig. 3) constitutes further evidence that the damage caused by concentrated contrast media results from other than simple osmotic effects. The diminished red blood cell survival illustrated in the lower curve on Figure 6 further documents the extent of the red blood cell damage; and provides an interesting avenue for further investigation into the cause for normal *in vivo* survival of a small proportion of the injected cells. Other observations which merit further study are the discrepancies in hemolytic potential among different contrast media (illustrated in Fig. 3) and the suggestion that hemolytic potential

may be decreased by employing "combined" contrast media (*e.g.*, ditriokon, hypaque, "triple medium") in which the total iodine content is high but in which no single organic iodide is present in high concentration. The importance of the stability of organic iodide "combinations" deserves special emphasis. For example, the "triple medium," which caused relatively little hemolysis when freshly prepared (Fig. 3), was noted to darken in color during 3 weeks' storage at summer room temperatures and when retested proved to be almost as hemolytic as the 70 per cent urokon over which it had excelled previously.

It remains an open question whether serious reactions to roentgenologic contrast media may result from aggregation of red blood cells, with secondary vascular plugging, or from a direct toxic effect of the media upon the vessel wall itself, particularly upon the endothelium. Mudd and his co-workers[12] have demonstrated what they describe as "agglutinative thrombosis" within small pulmonary vessels in stained sections of lung tissue obtained from dogs within 1 to 60 minutes after injection of 70 per cent urokon into the pulmonary artery. Similar findings have been described by Read and co-workers,[14] who made direct observations of small pial and mesenteric vessels following the rapid intra-arterial injection of hypertonic solutions (20 per cent NaCl, 50 per cent glucose). In another report, Read[13] has stressed the important role of the red blood cell in causation of the pulmonary hypertension that is observed following injection of concentrated contrast media into the pulmonary artery; for when the isolated lung was perfused with contrast media alone, elevation of pulmonary arterial pressure did not occur.

The present authors do not deny the possibility that intravascular aggregation of red blood cells may be important as a cause of clinical reactions. However, we were impressed by the fact that: (1) red cell clumping did not occur as long as the blood and contrast media were kept in motion; (2) clumping might be expected to occur *secondary* to greatly retarded blood flow, as appeared to be the case in the studies on rabbit nictitating membrane; and (3) the red blood cell clumps did not resemble tightly adherent immune red blood cell agglutinates, but rather represented a tangential aggregation of cells which could be easily dispersed when the stagnant red blood cell suspension was put back in motion. Also impressive were the evidences for direct damage to the red blood cell: hemolysis far exceeding what would be expected from the hypertonicity of the media, and impaired viability of over half of the red blood cells recovered after *in vitro* exposure to contrast medium. It seems certain that the exposed vascular endothelium must undergo somewhat similar changes to those observed in the red blood cell, and it might be expected that the anatomy of the endothelium would make it more susceptible than the red blood cell to some of the damaging effects of the media. Evidence for direct vascular damage has been reported by Zinner and Gottlob[16] and evidence for altered vascular permeability has been presented by Broman and Olsson.[1,2]

Whatever may be the actual mechanism(s) underlying nonallergic clinical reactions to contrast media, the present studies indicate that the red blood cell may serve as a convenient model for describing some of the properties of existing and newly developed media. It remains to be seen whether evidence of damage to red blood cells will be paralleled by a high incidence of serious clinical reactions. However, it would seem to be worthwhile to attempt a correlation of *in vitro* red blood cell findings with large scale clinical trials as a further adjunct in the continuing development of safer roentgenologic procedures.

SUMMARY

1. Four roentgenologic contrast media were studied for their *in vitro* effects upon fresh normal human erythrocytes.

2. In the presence of high concentrations of contrast media, red blood cells underwent rapid shrinkage and exhibited a tendency to clump in loosely adherent aggregates. Aggregates could be easily dispersed by gently mixing, and initial red blood cell aggregation could be largely prevented by keeping the blood in motion following exposure to contrast media.

3. Rapid penetration of contrast medium across the red blood cell membrane was demonstrated employing I^{131}-tagged urokon.

4. Concentrated contrast media were capable of directly damaging red blood cells, as evidenced by (a) hemolysis in excess of that expected due to osmotic effects alone and (b) impaired *in vivo* survival of a proportion of red blood cells recovered after *in vitro* exposure to one of the media.

5. Observations of the small vessels in the rabbit nictitating membrane following intracarotid injection of 70 per cent urokon were inconclusive but did not provide evidence of primary intravascular clumping of erythrocytes.

6. The possible value of attempting to correlate the *in vitro* effects of contrast media upon red blood cells and the *in vivo* incidence of nonallergic clinical reactions is discussed.

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Special gratitude is due to Mrs. Rosemary Egan for her technical assistance throughout the course of these investigations, and to Dr. M. K. King and Dr. A. P. Ley for their aid in the studies of the rabbit nictitating membrane.

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EXPERIMENTAL STUDIES OF DITRIOKON TOXICITY*

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IN VIEW of the development of cardiovascular diagnostic procedures which utilize large volumes of concentrated angiographic agents, the availability of preparations with a high degree of radiopacity, low toxicity, and ease of rapid administration has become important. Factors involved in the suitability of such preparations include the immediate cardiorespiratory effects, renal and nervous system effects, and those effects due to such physicochemical properties as viscosity and osmolality.

The recent combination of sodium diprotrizoate and diatrizoate, marketed as ditriokon,* is an attempt to provide a preparation with a high degree of radiopacity and low viscosity. It seemed worthwhile to evaluate this preparation experimentally for possible utilization in forward angiographic procedures requiring large doses of radiopaque materials.

ACUTE ANIMAL TOXICITY STUDIES

A. MATERIALS AND METHODS

Mongrel dogs of both sexes weighing from 5.5 to 10.9 kg. were anesthetized with sodium pentobarbital. Aortic blood pressure was monitored by means of a catheter in the proximal descending thoracic aorta connected to a Statham P23D strain gauge and recorded on a Sanborn Twin-Viso recorder. Lead II of the electrocardiogram was also monitored on this recorder. Observations were made of the microcirculation of the bulbar conjunctiva, of the presence and duration of apnea following injection, and of survival.

Ditriokon† was administered in graded doses from 3 to 7 cc. per kg. body weight, through a No. 280 polyethylene tube inserted into the vena cava via the femoral vein. Injection was made with the aid of a steel syringe and a hand lever injector, and in all instances the time required for injection was less than 1.5 seconds. Three dogs were injected at each dose level. Animals which survived for twenty minutes with normal vital signs were sacrificed, and, together with all animals which succumbed, were autopsied.

B. RESULTS

A summary of the results of the acute animal toxicity studies is presented in Table 1. In general, doses of 3 and 4 cc. of ditriokon per kg. body weight were well tolerated. There were some ST and T wave changes on the electrocardiogram but these were, in general, rapidly reversed. In a number of these animals, there was a short period of initial hypertension, uniformly followed by a period of hypotension. At the 3 cc. per kg. dose, the maximum systemic diastolic blood pressure drop ranged from 33 per cent to 50 per cent of the pre-injection values. At 4 cc. per kg., the maximum systemic diastolic blood pressure drop ranged from 70 to 91 per cent of the control values.

As the dose of ditriokon was increased to 5, 6, and 7 cc. per kg., the electrocardiographic and blood pressure changes became more severe. The presence of an atrial T wave, and severe S-T changes were accompanied by marked and irregular bradycardia, apparently of sinus origin. The post-injection phase of hypotension was uniformly profound (see Table 1).

Observations of the small blood vessels

* Mallinckrodt Chemical Works.

† Generously supplied by Mallinckrodt Chemical Works, St. Louis, Missouri.

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This study was supported in part by Grant H.-4335, National Heart Institute, United States Public Health Service.
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TABLE I

SUMMARY OF ACUTE ANIMAL TOXICITY DATA FOLLOWING MASSIVE INTRAVENOUS DOSES OF DITRIOKON

Dose (cc./kg.)	Weight (kg.)	Survival	Electrocardiographic Analysis			Blood Pressure Change		Pulse Pressure		Maximum Diastolic Blood Pressure Drop (%)	Red Blood Cell Aggregation	Pulmonary Pathology			
			Rhythm	Rate	Ischemic Changes	<5 sec.	>5 sec.	Initial	Max. Recovery	$\frac{-\Delta P_D}{P_D} \times 100$	Bulbar Conjunctiva	Hemorrhage	Edema	Dilated Capillaries	Dilated Veins
3	7.3	Yes	Reg.	—	3	Incr.	Decr.	25	40	48	2+	2+	—	2+	2+
	5.4	Yes	Reg.	—	2, 3	Incr.	Decr.	40	50	33	0	—	—	1+	—
	9.6	Yes	Reg.	—	3	—	Decr.	30	50	50	2+	—	—	2+	—
4	8.2	Yes	Irreg.	Decr.	2, 3	Incr.	Decr.	45	110	70	2+	1+	1+	1+	—
	10.9	Yes	Irreg.*	Decr.	1, 2, 3	Decr.	Decr.	40	55	91	2+	—	—	2+	—
	9.1	Yes	Reg.	Decr.	1, 2, 3	Decr.	Decr.	25	50	75	3+	—	—	—	1+
5	8.7	Yes	Irreg.*	Decr.	1, 2, 3	Decr.	Decr.	55	90	95	+	—	—	2+	—
	5.5	Yes	Reg.	Decr.	1, 2, 3	Decr.	Decr.	35	40	95	+	—	—	2+	—
	8.2	Yes	Reg.	—	2, 3	Decr.	Decr.	35	65	88	2+	1+	—	1+	—
6	5.5	Yes	Reg.	Decr.	2, 3	Decr.	Decr.	70	75	78	+	1+	—	2+	2+
	10.5	No	Irreg.*	Decr.	2, 3	Decr.	Decr.	30	—	100	3+	1+	—	4+	4+
	9.6	Yes	Irreg.	Decr.	1, 2, 3	Decr.	Decr.	45	70	83	3+	1+	—	1+	—
7	7.3	No	Irreg.	Decr.	2, 3	Decr.	Decr.	60	—	100	4+	2+	2+	1+	3+
	7.3	No.	Reg.	Decr.	2, 3	Decr.	Decr.	40	—	100	4+	2+	1+	3+	3+
	6.4	No	Irreg.	Decr.	1, 2, 3	Decr.	Decr.	35	—	100	4+	3+	3+	2+	2+

* Dropped beats.
1—Atrial T wave.
2—ST depression.
3—T wave elevation.

of the bulbar conjunctiva revealed the presence of some red blood cell aggregation in almost all of the dogs following ditriokon injection. In general, there was some correlation between the dose of ditriokon administered and the degree of aggregation visualized.

In the dogs receiving 3 cc. of ditriokon per kg., there was little or no respiratory arrest following injection (Table 1). This is in marked contrast to the normal 30 to 40 second period of apnea which follows the injection of an equal volume of hypaque 90 per cent in the dog.[1] With larger injections of ditriokon, apnea appeared and became progressively longer with increased dosage.

At autopsy, in the 3 and 4 cc. per kg. dose range, most animals had minimal areas of pulmonary hemorrhage, or dilatation of pulmonary capillaries and/or veins. As the dosage of ditriokon was increased, the appearance of localized areas of pulmonary hemorrhage was common. However, dilated pulmonary capillaries and veins were the most prominent and consistent histologic findings at all dose levels tested. Histologic sections of the heart, liver, pancreas, spleen, small intestine, and kidneys of all of the animals studied failed to reveal any other significant pathology.

A comparison of the mortality subsequent to massive intravenous doses of ditriokon and hypaque 90 per cent (as reported previously[1]) is shown in Figure 1.

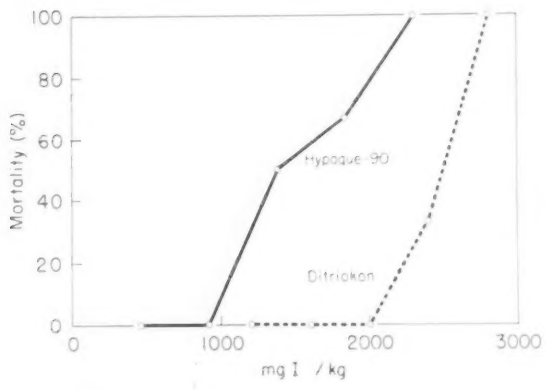

FIG. 1. Dose-mortality curve following rapid intravenous injections of ditriokon (40 mg. I/cc.) and hypaque 90 per cent (46 mg. I/cc.) in the dog.

It may be noted that mortality has been plotted against milligrams of iodine delivered per kg. body weight, rather than in terms of volume of media. It is felt that this is a more equitable way of comparing opacification ability with mortality.

Several examples of the blood pressure and electrocardiographic response to massive doses of ditriokon are shown in Figures 2, 3, and 4. At a dose of 3 cc. per kg., it may be seen that there are essentially no changes in cardiac rate or rhythm, and minor and rapidly reversible T wave changes in the electrocardiogram (Fig. 2). When the dose is increased to 6 cc. per kg., an initial hypertension, accompanied by prompt S-T segment changes, is followed by a severe sinus bradycardia, profound hypotension, and death (Fig. 3). A similar pattern, shown in Figure 4, was recorded in an animal receiving 7 cc. of ditriokon per kg. Electrocardiographic changes in the animals receiving very large doses of ditriokon are seen within 1–2 seconds of the start of injection. Some direct toxic action or reflex mechanism involving the sino-atrial node appears to be involved.

PHYSICOCHEMICAL STUDIES

A. OSMOLALITY

Determinations of the osmolality of a number of commonly employed angiographic media were performed using a Fiske osmometer,* which employs the freezing point depression principle. The studies are summarized in Table II, in comparison to normal blood osmolality, and indicate the relative hypertonicity of the substances studied. There is a good correlation between the osmolality and the amount of iodine in the compounds studied.

These studies are of particular interest in view of the recent report which implicates osmolality as one of the most important factors in the subsequent development of red blood cell aggregation and hemodynamic alterations following the rapid injection of these agents.[3] Ditriokon

* Through the cooperation of Dr. Theodore R. Grage.

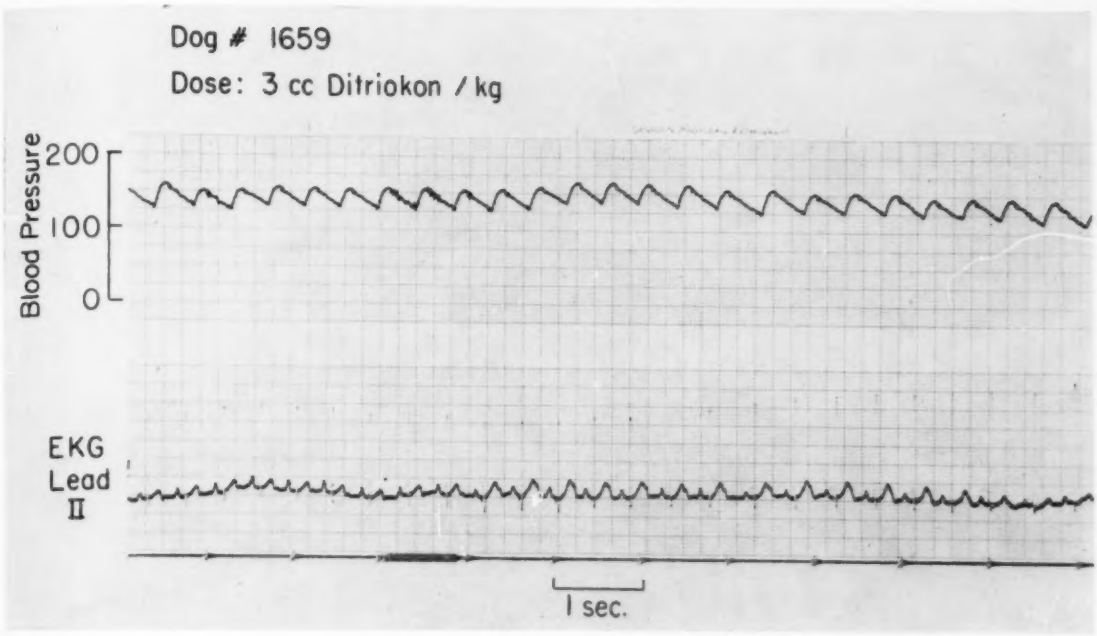

Fig. 2. Blood pressure and electrocardiographic response to the rapid intravenous injection of 3 cc./kg. ditriokon. Some ST segment and T wave changes are seen, along with a minor blood pressure depression. Injection is indicated on the timer tracing.

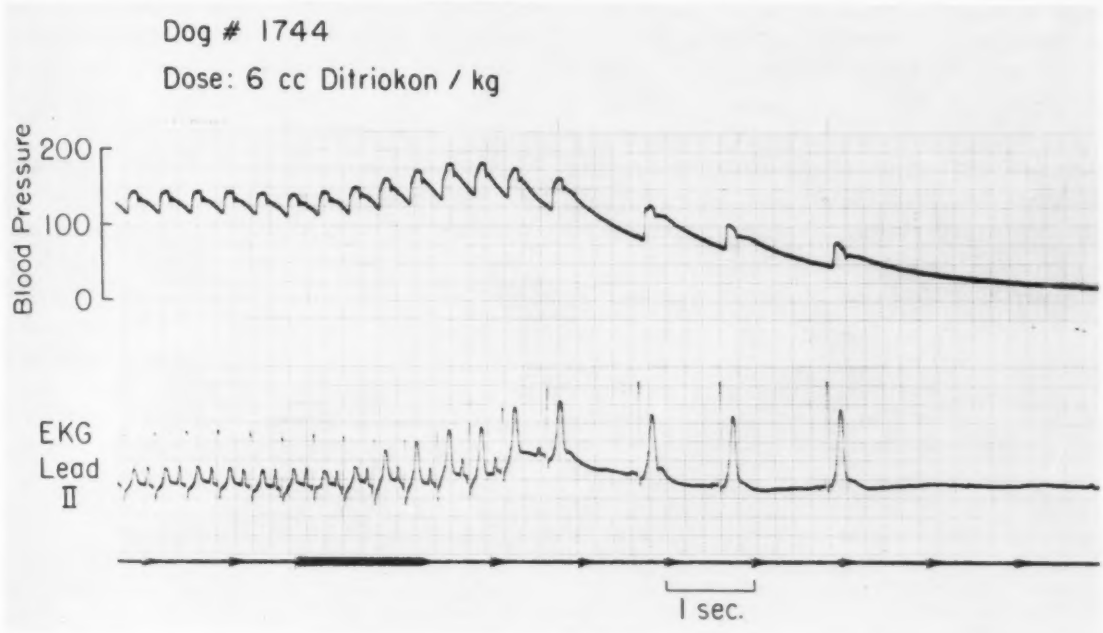

Fig. 3. Marked response to a 6 cc./kg. dose of ditriokon with some hypertension followed by profound hypotension and bradycardia. Note the almost immediate electrocardiographic changes, which begin within one second from the start of the injection (timer line).

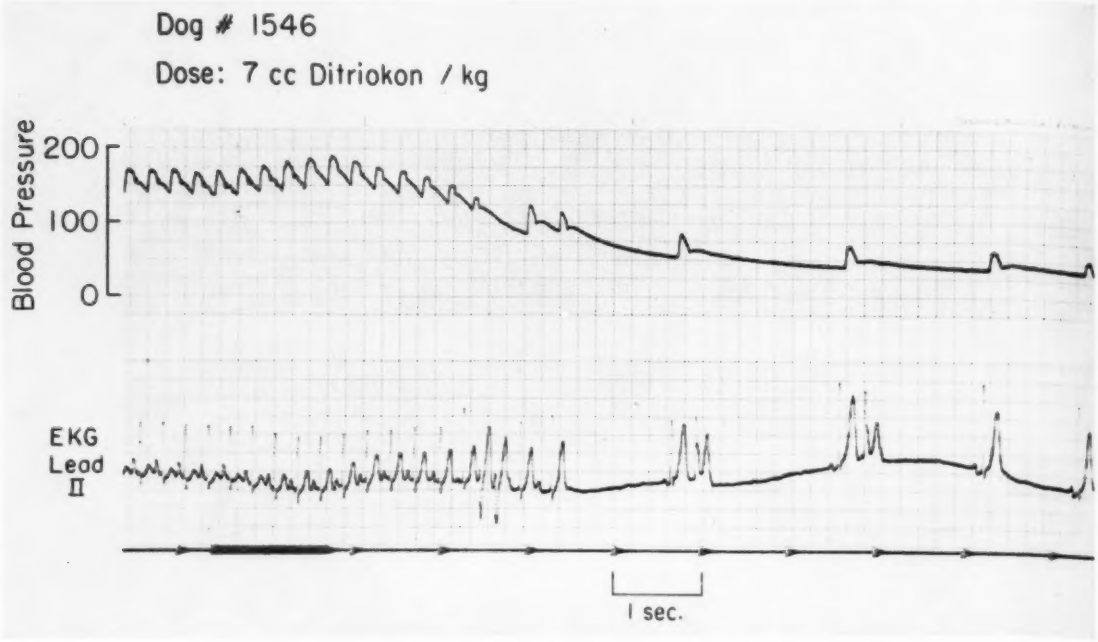

FIG. 4. An intravenous dose of 7 cc./kg. ditriokon was uniformly lethal. Marked electrocardiographic aberrations accompany the profound and rapidly fatal hypotension.

is essentially equal in osmolality to hypaque 90 per cent, and yet, utilizing identical injection procedures, it appears to have an LD_{50} approximately twice that of hypaque 90 per cent.[1] In this regard, it should be noted that the ditriokon injections were considerably more rapid than those of hypaque 90 per cent, as a result of its decreased viscosity, although the same injection apparatus was used in both studies. This factor was expected to increase its toxic effects, but despite this, the observed mortality was decreased.

B. VISCOSITY

Utilizing a modified Eckstein, Book and Gregg viscosimeter, measurements of the viscosity of a number of commonly utilized

TABLE II

OSMOLALITY AND IODINE CONTENT OF ANGIOGRAPHIC AGENTS

Preparation	Osmolality (mOsm./L.)	Per Cent Iodine (gm./100 cc.)	Ratio (osmolality/per cent I)
Renografin 60%	1360	29.3	46.5
Hypaque 50%	1376	30	45.9
Renografin 76%	1648	37	44.6
Hypaque 75%	1716	39.3	43.8
Cardiografin 85%	1900	40	47.5
Ditriokon 68.1%	1904	40	47.6
Hypaque 90%	2020	46	44.0
Urokon 70%	2140	46	46.5
Whole Blood	285	—	—
			Average: 45.8
			Range: 43.8–47.6

angiographic preparations were performed through a temperature range of approximately 0–50 C. The preparations studied included 50, 75 and 90 per cent hypaque,* 60 and 76 per cent renografin,† 85 per cent cardiografin† and ditriokon.

The results of these measurements are presented in Figure 5, in comparison with the viscosity of whole blood. It may be noted that ditriokon is considerably less viscous than the other tested agents with comparable iodine contents. In addition, it may be noted that heating this agent from room temperature (25°C.) to body temperature (37°C.) has little effect upon its viscosity, in contrast to the marked effects of heating some of the more concentrated diatrizoate preparations.

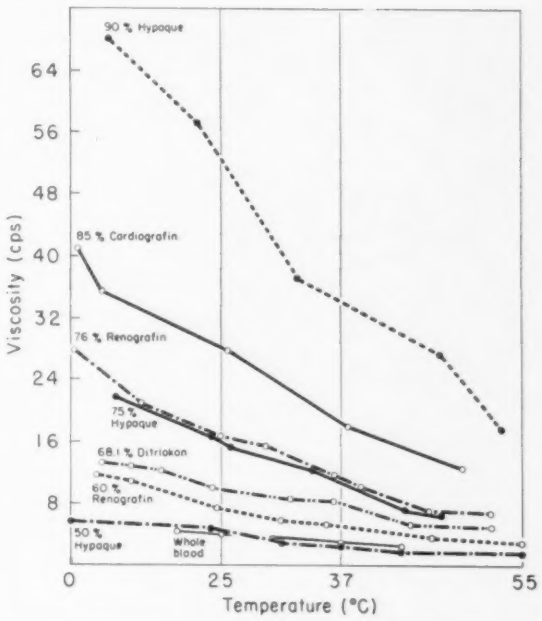

Fig. 5. Viscosity of commonly employed iodinated radiopaque compounds used for angiography over a temperature range from 0–50° C.

c. Comparative Injection Rates

Utilizing the information obtained from the viscosity measurements and the percentage of iodine contained in the various preparations, calculations of the comparative injection rates of (a) the volume of material and (b) the amount of iodine which can be injected with an identical injection pressure, catheter system, etc., have been made. For this purpose, the viscosity of the substances at room temperature has been utilized. It may be noted that more than 5 times as much ditriokon as hypaque 90 per cent could be injected in a given period of time, and this would contain 4.5 times as much iodine. These calculations assume laminar flow, which is a valid assumption only in low pressure injection systems. In high pressure injections, flow is often turbulent, and the differences between injection rates are not as marked as those calculated in Table III. In the acute animal toxicity studies, the ratio of the rates of injection of ditriokon and hypaque 90 per cent varied from 2 to 6.

Discussion

The animal toxicity studies indicate that massive doses of ditriokon may be given with considerably greater initial safety than equal doses of hypaque 90 per cent. The value of these observations for such studies as intravenous aortography appears obvious. While the clinical application of such large doses as have been used in these studies is not immediately justifiable, the utilization of ditriokon for such studies should be associated with increased patient safety, and some cautious increase in patient dosage may be justified.

The low viscosity of ditriokon permits

Table III

Calculated Comparative Injection Rates (per unit time)*

Preparation	Volume	Amount of Iodine
Hypaque 90%	1.0	1.0
Cardiografin 85%	1.8	1.6
Renografin 76%	3.1	2.5
Hypaque 75%	3.3	2.7
Ditriokon 68.1%	5.2	4.5

* Preparation at room temperature (25°C.), with standard injection pressure, catheter system, etc., and relative to hypaque 90 per cent as unity, based on viscosity measurements, and assuming laminar flow (see text).

* Generously supplied by Winthrop Laboratories, Inc.
† Generously supplied by E. R. Squibb & Sons.

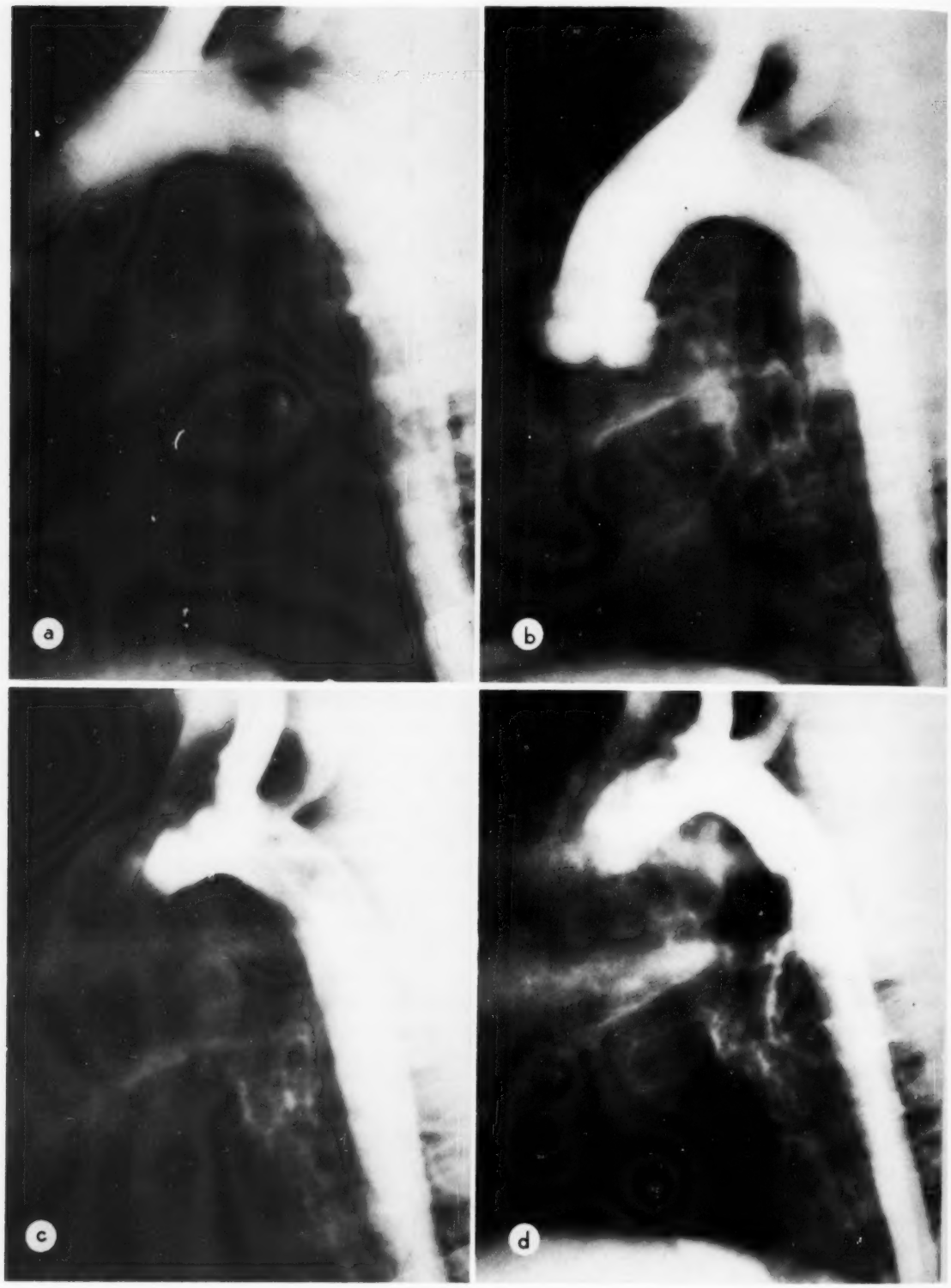

FIG. 6. Comparison of thoracic aortograms of the same dog, utilizing identical injection apparatus and pressure, and equal volumes of (*a*) hypaque 90 per cent, (*b*) ditriokon, (*c*) hypaque 75 per cent and (*d*) cardiografin 85 per cent.

its utilization at room temperature, as well as its more rapid injection under standard injection conditions. The marked decrease in cardiovascular reaction to this hypertonic agent, as compared with hypaque 90 per cent, is of considerable theoretic significance in any attempt to invoke hypertonicity alone as the cause of the hemodynamic alterations observed. The data presented appear to show that viscosity has a significant role in this mechanism.

While these studies indicate that ditriokon is relatively well-tolerated insofar as the acute cardiorespiratory response to rapid intravenous injection is concerned, it must be noted that the sodium diprotrizoate (miokon) fraction has been demonstrated to contain the potential for nephro-and neurotoxicity when administered separately. Long-term studies of the toxic potential of ditriokon to the kidney and the central nervous system therefore appear appropriate for future investigation.

SUMMARY

Experimental studies have been performed in an attempt to evaluate a new angiographic preparation. Acute intravenous toxicity studies in the dog indicate that the LD_{50} of ditriokon is approximately twice that of hypaque 90 per cent. The ratio of osmolality to percentage of iodine content is essentially constant for all the angiographic preparations studied. Viscosity studies indicate the capability of ditriokon to be delivered in large amounts per unit time (with high iodine content) under standard injection conditions. Ditriokon would appear worthy of clinical trial in procedures requiring large amounts of concentrated radiopaque angiographic media. Caution is advised, however, inasmuch as the potential of ditriokon to cause central nervous system and renal damage has not been evaluated in long-term experiments.

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THE ROENTGEN DIAGNOSIS OF HEMANGIOMA OF BONE*

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HEMANGIOMA of bone is a benign, slowly growing tumor arising from newly formed blood vessels and it is one of the primary osseous neoplasms. Histologically, two types are recognized, cavernous and capillary. The cavernous is made up of large, thin-walled vessels and sinuses which are lined by a single layer of endothelial cells interspersed among resorbed bony trabeculae. The capillary type is composed of fine capillary loops which tend to spread outward in a sunburst fashion. Cavernous hemangiomas are commonly found in the vertebrae and skull, while the capillary variety is more likely to be encountered in flat bones and in the metaphyses of the long bones.[50]

Osseous hemangiomas are rather rare tumors. The first report referring to the roentgen findings in this tumor was by Hitzrot[27] in 1917. Bucy and Capp[10] in 1930, described the appearances in 7 cases selected from the Bone Tumor Registry. Lichtenstein[39] has observed only a few of these neoplasms as surgical specimens. At the Mayo Clinic, Dahlin[17] encountered 13 hemangiomas of bone constituting 0.6 per cent of the total primary bone tumors at that institution. As Jaffe[30] has stated, when one excludes both the vertebral hemangiomas and those in the skull, then this type of primary neoplasm becomes an uncommon condition indeed.

A review of the material available to us brought to light 45 cases of primary hemangioma of bone, all of which were histologically proved except for a few having a characteristic roentgen pattern in the vertebrae and skull. The present purpose is to report the roentgen appearance of these tumors with the object of making their identification more reliable for the roentgenologist.

The location of the 45 hemangiomas is given in Table I.

TABLE I

LOCATION OF 45 HEMANGIOMAS

Spine				Facial Bones	
	Cervical	1		Mandible	3
	Thoracic	5		Nasal	1
	Lumbar	8		Zygoma	1
			Ribs		5
Skull		13	Clavicle		1
			Foot		2
			Long Bones		5

More than half of the hemangiomas were in the skull and spine. An inflated percentage of hemangioma in the other bones may be present because of the tendency of patients with tumors and tumor-like lesions of the bone to find their way to this institution plus the fact that a number of spinal tumors, having been discovered incidentally, undoubtedly escaped being recorded in the diagnostic files.

HEMANGIOMA OF THE SPINE

Because of a general familiarity on the part of radiologists with the appearance of hemangioma in this site, only a summary of the material will be given. Apparently there is a wide disparity between the anatomic and the roentgen incidence of vertebral hemangioma. This fact has been demonstrated by Schmorl,[52] Töpfer[58] and later by Junghanns.[35] In 3,829 vertebral columns studied in Schmorl's institute, the incidence of hemangioma was revealed to be 10.7 per cent, and another report of

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

10,000 spinal columns studied revealed a similar incidence. It is now well known that the smaller, asymptomatic vertebral hemangiomas rarely can be demonstrated on roentgenograms, a fact proved by roentgen examination of autopsy material.

Hemangioma of the spine usually is a single lesion, but sometimes two or more vertebral bodies are involved. While the process is generally in the vertebral body, other portions of the vertebra can be the site of the disease. When the hemangioma is of adequate size, the roentgen diagnosis is usually not difficult.

There may be a slight general diminution in the over-all bone density of the vertebral body. The most characteristic finding, however, is the presence of parallel, somewhat fuzzy, vertical linear streaks, which give the so-called "corduroy cloth" appearance. This effect is noted particularly in the lateral projection, and it is sometimes accompanied, particularly in sagittal projection, by a fine reticulated or "honeycomb" texture. The cortex of the vertebral body may become somewhat fuzzy, ragged and ill-defined, but usually its outer border is intact (Fig. 1, *A* and *B*; and 2, *A* and *B*). With compression fracture, many of these characteristic findings become less evident or are obscured.

A compression fracture was observed in 3 instances. One, in which the compression of the body was only partial, showed that many of the roentgen characteristics of hemangioma were preserved (Fig. 3, *A* and *B*). In the second, the gradual progression of the compression was followed by serial roentgen studies (Fig. 4, *A*, *B* and *C*). The third revealed extensive destruction and such a pronounced degree of collapse of the vertebral body that roentgenologically a diagnosis of hemangioma could not possibly be made.

Most hemangiomas of the spine are asymptomatic. However, in 2 of the patients, there was a vague intermittent type of pain. In the 3 instances where compression fracture occurred, there were both subjective and objective findings. Two of the patients complained of severe pain in the back and examination disclosed spasm

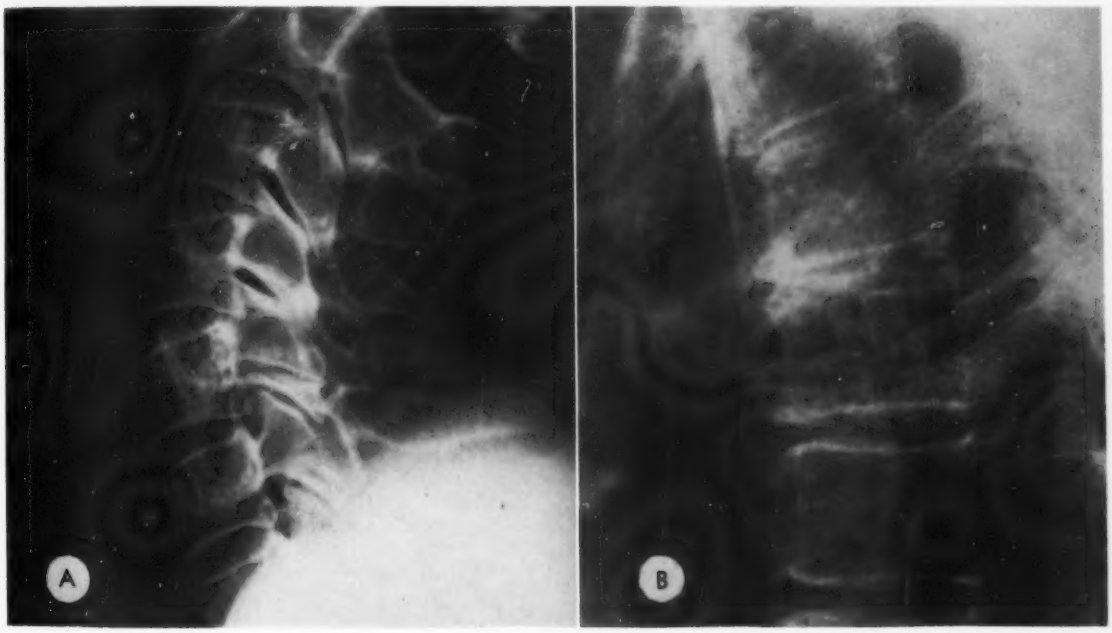

Fig. 1. (*A*) Hemangioma of the fifth cervical vertebra showing involvement of the body, pedicles and spinous process. (*B*) Hemangioma of the sixth thoracic vertebra. Note the pillar-like trabeculae running in a longitudinal axis.

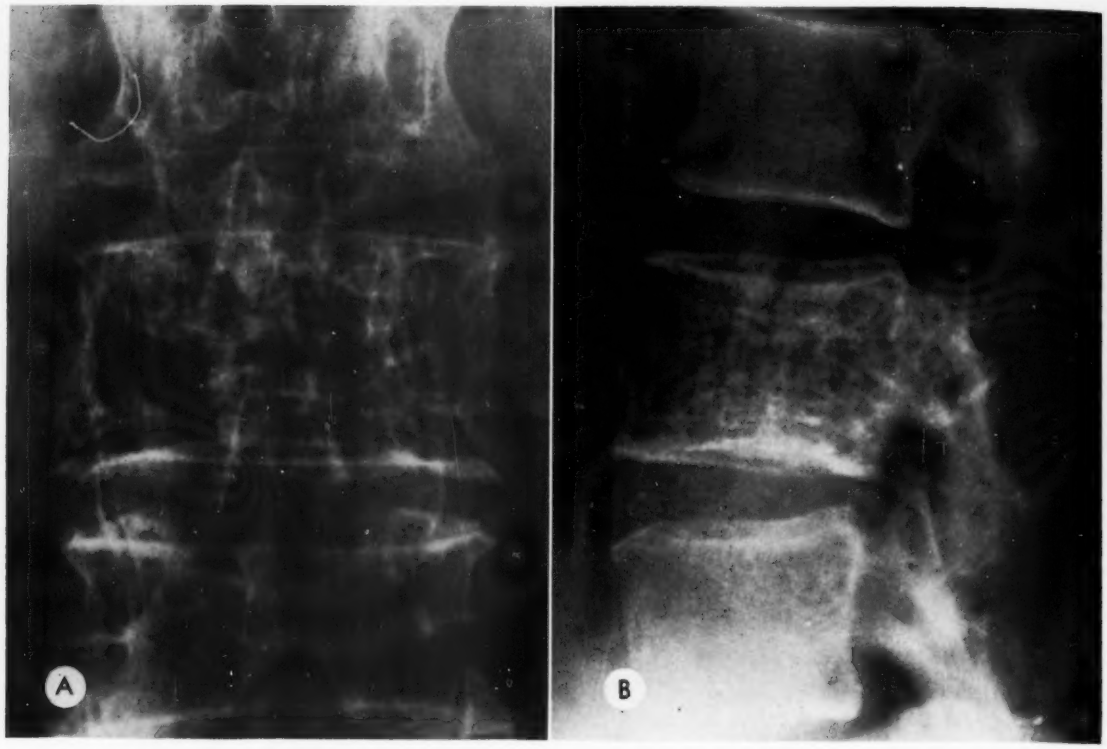

FIG. 2. (*A* and *B*) Hemangioma of the third lumbar vertebra. The parallel linear streaks running vertically in the body give a "corduroy cloth" appearance.

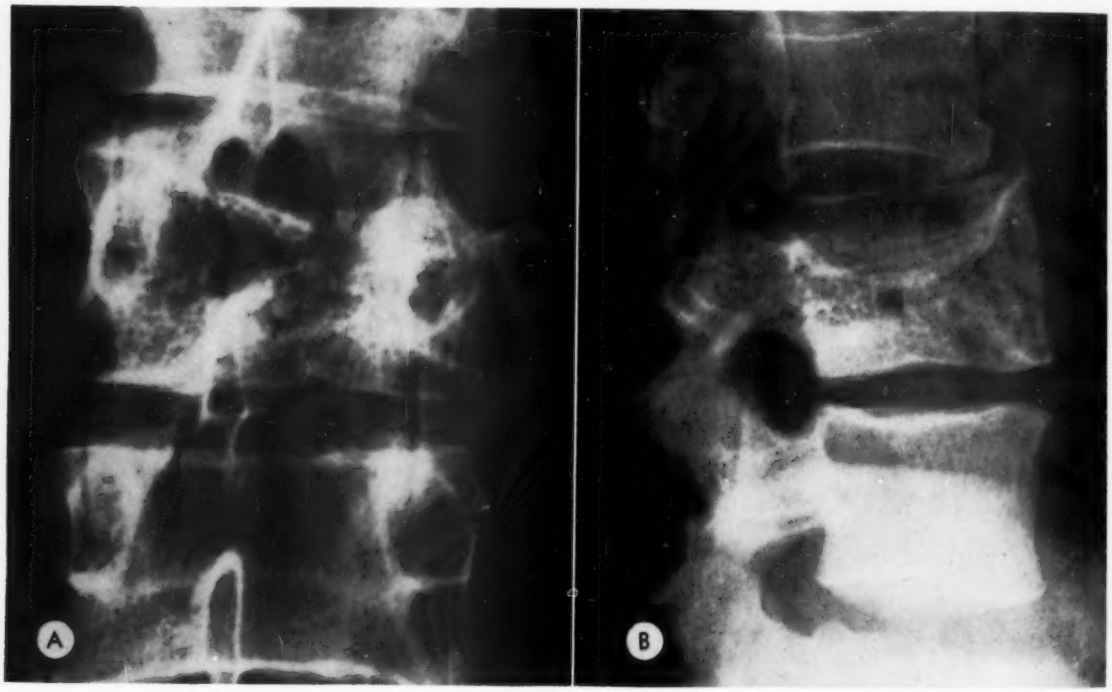

FIG. 3. (*A* and *B*) Hemangioma of the third lumbar vertebra complicated by a compression fracture. The characteristic vertical striations have been replaced by a coarse, irregular trabecular pattern.

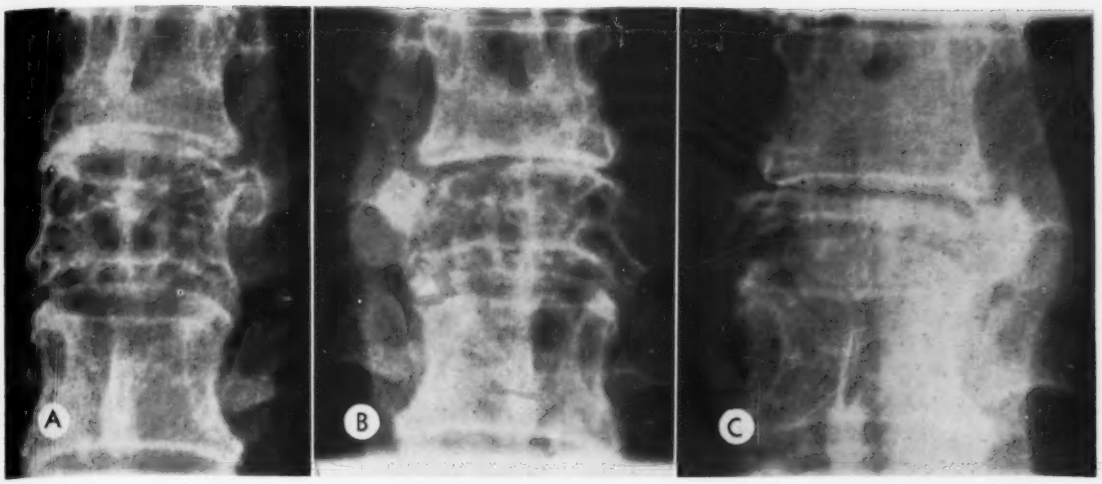

FIG. 4. (*A*, *B* and *C*) Hemangioma of the ninth thoracic vertebra showing interval compression over a period of ten weeks.

and rigidity of the muscles of the lumbar region. The third displayed signs of compression of the spinal cord as evidenced by spastic paraplegia with a sensory level at the thoracic segment. Four of the patients were treated by irradiation and showed a satisfactory response. Two were relieved of their complaints, and they have remained symptom-free for more than ten years.

Schlezinger and Ungar[51] reviewed the literature in 1939 and added 2 cases of their own to make a total of 40 patients who had neurologic changes associated with compression of the spinal cord due to hemangioma of bone. Robbins and Fountain[49] recently reported a successfully treated case of hemangioma of the cervical vertebrae with severe myelocompression, stating that it was the eleventh such case described.

In the differential diagnosis, hemangioma of the vertebrae sometimes must be distinguished from Paget's disease. In osteitis deformans there is usually a more uniform increase in bone density and there is framing of the vertebral body by a zone of cortical thickening and sclerosis. Malignant lymphomas occasionally show changes similar to hemangioma of bone and metastatic cancer and multiple myeloma similarly must be considered in differential diagnosis.

HEMANGIOMA OF THE SKULL

The first such case was reported by Toynbee in 1845. In 1949, Wyke[60] reviewed the literature and uncovered 39 primary angiomatous lesions in the skull.

The hemangiomas of the skull reported here are confined to the frontal bone. The lesion tends to be quite typical roentgenologically, being represented by a single area of rarefaction, which is usually either round or oval in shape. There is a variation in size from 1 to 7 cm. in diameter. The internal pattern is characterized by fine, delicate striae or reticulations, which being closely knit often assume a "honeycomb" appearance (Fig. 5, *A* and *B;* and 6). These striations on occasion are exaggerated and give a "sunburst" appearance.

A few of the smaller lesions seemed almost entirely lytic with no discernible internal pattern. The margins of the defects were sharp and well delineated from the surrounding normal bone and sometimes a fine margin of bone condensation was seen at the periphery.

Several cases were somewhat atypical. One was spindle-shaped, measuring 7.0×2.5 cm. Another of the larger lesions, measuring 5.5×5.0 cm., showed a centrally placed radiolucency surrounded by the usual

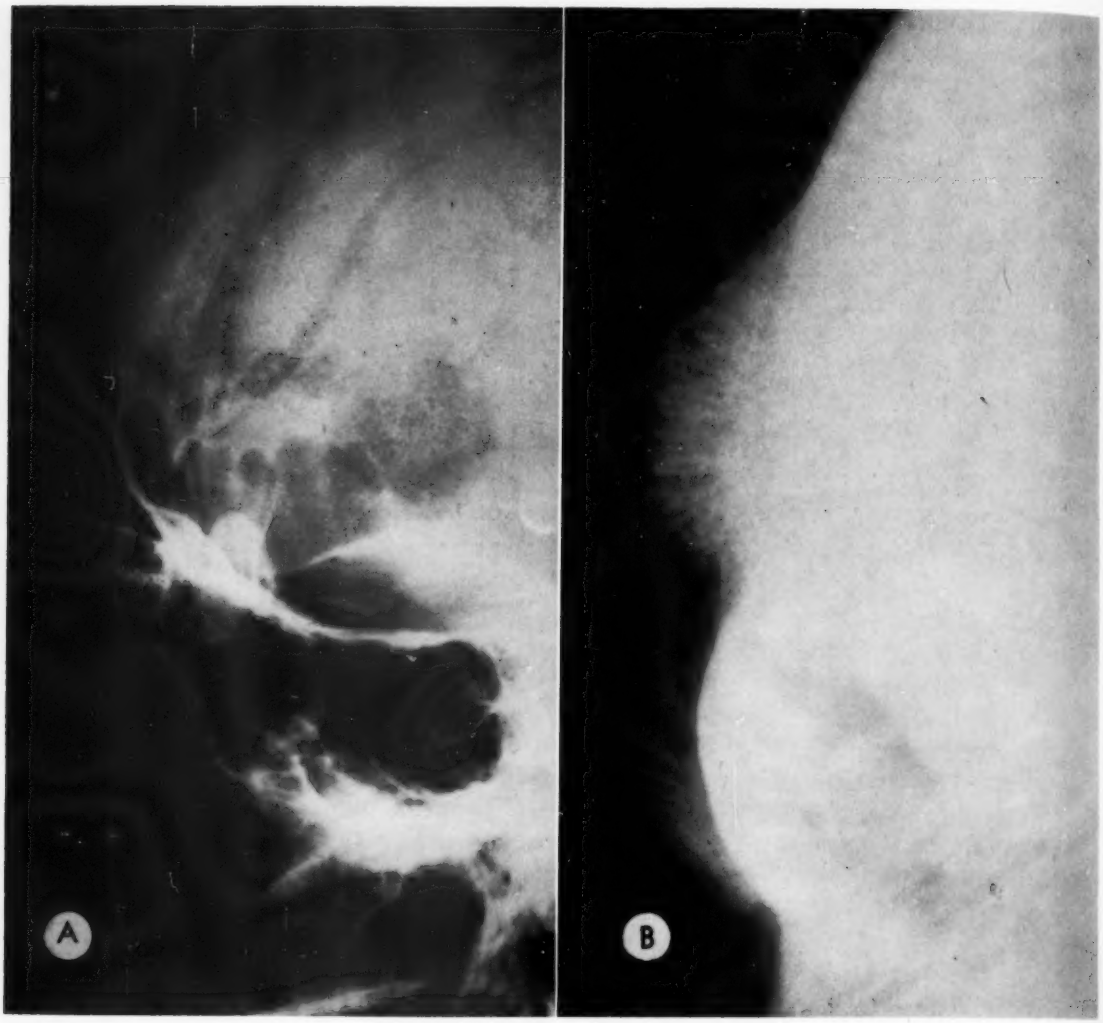

FIG. 5. Hemangioma of the skull. (*A*) A round area in the frontal bone showing a finely porous "honeycomb" rarefaction. (*B*) Tangential view of same skull showing radial striations giving the "sunray" type of appearance.

"honeycomb" type of septation, while at the periphery of the lesion there were clustered small, round, fairly distinct radiolucent areas. A hemangioma measuring 3×3 cm. and seen as a round defect in the right frontal bone had a dense compact segment of bone in its medial portion. However, there was an accompanying "honeycomb" pattern surrounding the dense central area. One hemangioma was purely lytic in one half of the lesion, whereas the other half presented the characteristic "honeycomb" reticulation.

Tangential or profile views are particularly informative and constitute a necessary part of the roentgen examination of lesions in this area. Ordinarily, the bony tumefaction is seen extending outwards beyond the normal confines of the bone. This external bulging is sometimes accompanied by an erosion of the outer table of the skull, whereas the inner table tends to be well maintained. The profile view frequently discloses vertical striations assuming a "sunray" type of appearance. These bony striae seem to be directed in a radial

manner as though originating from a common center (Fig. 5*B*). Six of the cases presented this appearance.

Conditions to be considered in differential diagnosis include cancer metastases and multiple myeloma, and, when the profile views reveal vertical striations, a meningioma may have to be ruled out. A few hemangiomas bear a close resemblance to epidermoids. However, the outline of these lesions tends to be more serpiginous, the margins are fairly dense due to a fine sclerotic border, and they do not have a reticulated or "honeycomb" pattern. Osteoma at times closely resembles hemangioma and in some instances the differential diagnosis is almost impossible to make with assurance. Generally speaking, however, an osteoma is uniformly dense or has a pattern that looks more like normal trabeculae.

HEMANGIOMA OF THE MANDIBLE

Two of the 3 cases in the mandible presented a fairly classic roentgen appearance. One was a nineteen year old female who had noted a swelling two years previously. This gradually increased in size.

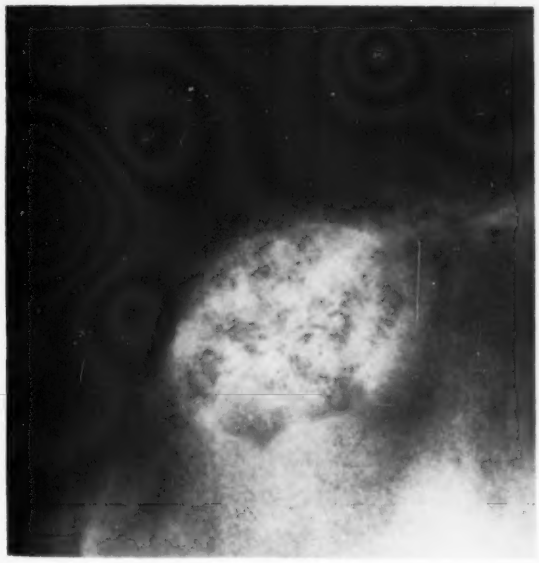

Fig. 6. Hemangioma of the skull showing a more coarse "honeycomb" rarefaction.

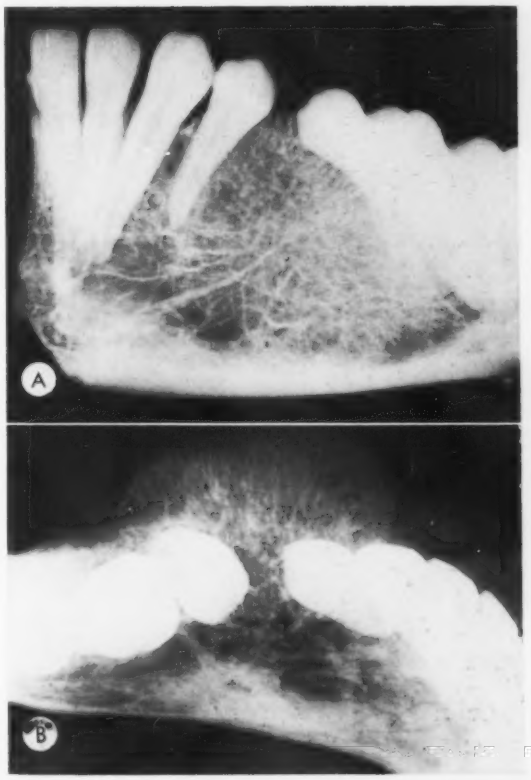

Fig. 7. Hemangioma of the body of the mandible. (*A*) A roentgenogram of the gross specimen showing the classic "sunray" appearance. (*B*) Tangential view showing the characteristic radiating spicules at right angles to the bone.

Physical examination showed a hard enlargement, 4×3 cm., which involved the entire thickness of the bone. Roentgen examination demonstrated a multiloculated expanding lesion in the body of the left side of the mandible. The expanded area contained thick coarse trabeculations radiating outwards from a somewhat ill-defined center point. The cortex was intact and there was no evidence of any soft tissue component. The pathologic diagnosis from an incisional biopsy was hemangioma of bone, cavernous type.

A roentgenogram of the second case revealed an expanding, osteolytic lesion with numerous trabeculae radiating in all directions. Tangential views displayed characteristic radiating spicules at right angles to the surface of the bone. A roentgenogram

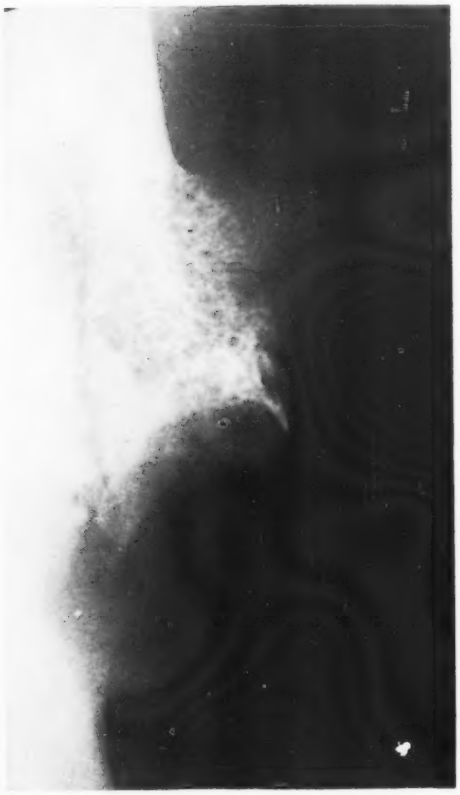

FIG. 8. Hemangioma of the nasal bones. The internal pattern of the expanding lytic lesion is that of a fine fibrillary network of bony striae.

of the resected gross specimen showed the classic "sunray" appearance (Fig. 7, *A* and *B*).

Castleman[12] discussed a case of hemangioma in the mandible from the files of the Massachusetts General Hospital, in which the roentgenograms showed an expansion of the bone, thinning of the cortex and an irregular, fine lacy osseous network producing honeycombing.

HEMANGIOMA OF THE NASAL BONES

The first case of primary hemangioma of the nasal bones was reported in 1936 by Neivert and Bilchick.[42] Another was described by Bloom[8] in 1954. The roentgenograms of the presently reported case showed an expanding predominantly lytic lesion, ovoid in configuration. The cortex was completely gone, but there did appear to be a fine limiting shell of periosteal new bone. The internal pattern of the tumor created a fine fibrillary network, highly suggestive of the diagnosis of hemangioma (Fig. 8).

HEMANGIOMA OF THE ZYGOMA

This is the only case known in which hemangioma has been described in this bone.

The patient, a forty-six year old female, noted a swelling below the left eye for two years. Physical examination disclosed a bony protrusion 2.5 cm. in diameter along the inferior outer portion of the left orbit.

Roentgenographic examination demonstrated a somewhat expanding lytic lesion in the superior portion of the left zygoma. The inferior margin of the lesion was distinct and the internal pattern of the tu-

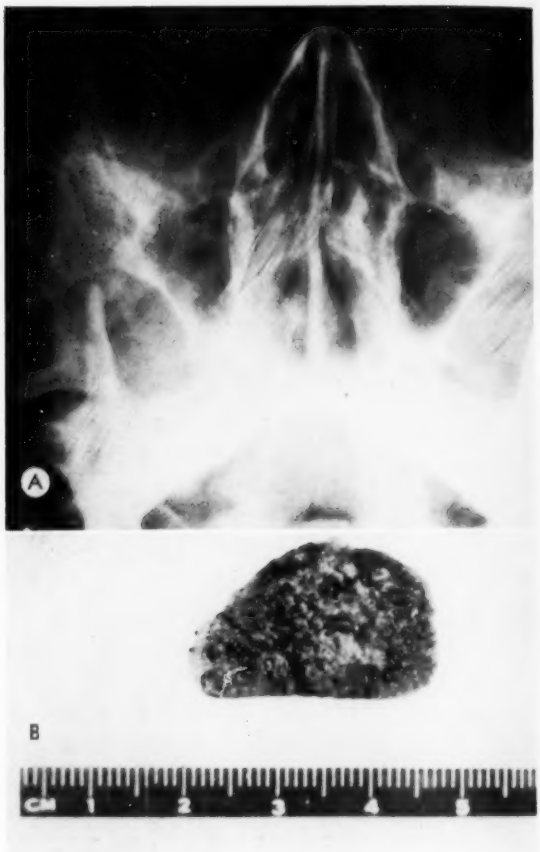

FIG. 9. Hemangioma of the right zygoma. (*A*) The interior of the tumor shows an exaggeration of the diploic spaces giving a "honeycomb" type of appearance. (*B*) Photograph of resected specimen.

mor had a reticulated and "honeycomb" appearance (Fig. 9, *A* and *B*). In general the roentgen appearance of the lesion was quite typical. The tumor was completely excised. The pathologic diagnosis was hemangioma of bone.

HEMANGIOMA OF THE RIBS AND CLAVICLE

The roentgen appearance of hemangiomas in these sites was sufficiently unique so as to offer special problems in differential diagnosis.

A thirteen year old white male with a rib lesion was struck in the right side of the chest two years previously, suffering sufficient pain to require strapping. Since that time, there had been intermittent discomfort. Physical examination disclosed tenderness, but no swelling.

Roentgen examination showed an expanding lytic lesion anteriorly in the right sixth rib. The bony architecture was completely lost in a portion of the defect. A very faint cortical rim could be detected about the periphery, forming a sharp line of demarcation with the normal bone. The rib was resected and the pathologic diagnosis was hemangioma. The roentgen appearance of this tumor can hardly be considered typical of hemangioma. Some resemblance to giant cell tumor, fibrous dysplasia and chondroma is noticeable.

A routine chest roentgenogram revealed a puzzling process in the left fifth rib of a twenty-nine year old female who was asymptomatic. The posterior portion of the rib was widened and replaced by a coarse web-like pattern of vacuolated appearance. The superior and inferior borders formed irregular, thinned-out cortical margins, and there were a few bony septa or ridges perpendicular to the axis of the rib. A surrounding soft tissue mass measuring about 6 cm. was present (Fig. 10, *A* and *B*). A resection of the fourth, fifth, sixth and seventh ribs was carried out and the histologic diagnosis was hemangioma of the bone. The roentgen appearance of this particular tumor can be confused with osteogenic sarcoma because of the irregular destruction of bone, the unusual internal pattern and the soft tissue mass. The significant point here is the fact that a soft tissue mass may occasionally accompany hemangioma of bone.

On a routine chest roentgenogram of a thirty-five year old asymptomatic female, a bizarre expanding defect of the right eighth rib was discovered. This portion of

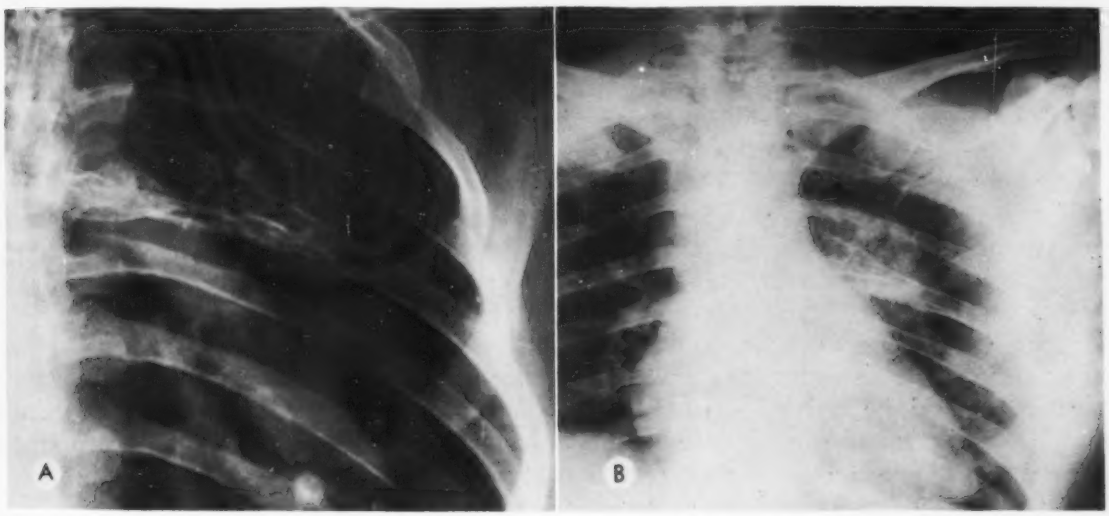

Fig. 10. Hemangioma of the left fifth rib. (*A*) The posterior portion of the rib is widened and replaced by coarse bony septa which give the bone a vacuolated appearance. (*B*) A chest roentgenogram showing a soft tissue mass surrounding the rib lesion.

FIG. 11. Hemangioma of the clavicle. (*A*) roentgenogram of the gross specimen showing an exaggeration of the diploic spaces giving the bone a coarsely porous "honeycomb" pattern. The cortex is gone and perpendicular spiculations can be seen along the upper and lower borders of the external end of the clavicle. (*B*) A photograph of the gross specimen showing a lobulated soft tissue angiomatous mass surrounding the bone tumor.

the rib was enlarged to about twice its normal diameter and it presented a multiloculated appearance. Lytic areas were surrounded by irregular formations of bone. Cortical margins were thinned-out and distorted, and some areas appeared to be destroyed, particularly along the inferior border. The right eighth rib was resected and the pathologic diagnosis was cavernous hemangioma.

Again a diagnosis of malignant tumor, particularly osteogenic sarcoma, would have to be considered although the fairly well formed locules, the quite sharp line of demarcation between the involved and the normal bone, and the absence of periosteal reaction would be points of some value against malignant tumor. Castleman[11] discussed a very similar case and stated that it was the only one he had ever seen in a rib.

A forty-four year old white male stated that a lump had been present on his left collar bone for about nine years, following an injury. There was some associated pain which had increased in time. Roentgen therapy was given with some relief of symptoms. Recent physical examination disclosed a fixed, nontender mass in the outer third of the clavicle, which measured 5 cm. in diameter (Fig. 11, *A* and *B*).

This particular case is unusual not only because of the clinical history but the fact that the roentgen appearance strongly suggested a malignant tumor, especially osteogenic sarcoma, except that the bone destruction in the spongiosa was quite orderly and the existing "honeycomb" pattern would not ordinarily be encountered in a malignant neoplasm.

Bucy and Capp[10] reported a somewhat similar appearing tumor at the sternal end of the clavicle.

HEMANGIOMA OF THE BONES OF THE FOOT

One of the 2 cases of tumors occurring in this area is of special interest.

The patient was a fifty-five year old white female who had fallen and injured her right foot fifteen years previously. Eight weeks after the injury she complained of pain and some swelling was then noted. The pain subsided, but the swelling persisted for about ten years or so, after which she thought that an increase in the size of the swelling had taken place. Physical examination revealed a bony hard mass on the dorsum of the foot.

Roentgen examination showed a somewhat rarefied expanding lesion involving the entire extent of the first metatarsal and the adjacent tarsal bones of the foot as well. There were innumerable small locules giving the involved bone a characteristic "soap-bubble" appearance. A tangential view showed numerous fine delicate septa tending to run perpendicular to the long axis of the metatarsal bone (Fig. 12). The lesion was removed and the pathologic diagnosis was hemangioma of bone.

HEMANGIOMA OF SOFT PARTS INVADING BONE

Twenty-three cases were studied roentgenologically in which patients with soft tissue hemangiomas were subjected to roentgen examination. In 15 of these there was roentgen evidence of bone involvement. In most instances the hemangioma of the soft parts harbored multiple, round discrete calcifications representing phleboliths, a finding not noted in primary hemangioma of bone.

Soft tissue hemangiomas may reach very large proportions without involving bone, but if the neighboring bone is affected, it it may be altered in various ways. The simplest type of secondary change is merely a smooth indentation or erosion of the cortex. This was noted in 3 of the 15 cases. Another group showed a triad of roentgen findings; *viz.*, enlargement of the bone, coarsening of the bony trabecular structure and thinning of the cortex. In other instances pressure-type deformities of the neighboring bones were encountered. There was one such instance in which the tibia and fibula showed a saucerized defect with anterior bowing secondary to the presence of a large hemangioma (Fig. 13). In addition, the bony texture was altered to a degree that suggested the presence of hemangioma within the bone itself.

Fig. 12. Hemangioma of the first metatarsal and adjacent tarsal bones of the foot. The presence of numerous small locules give the bone tumor a "soap-bubble" appearance. Note the numerous fine delicate septa running perpendicular to the long axis of the bone.

Fig. 13. Pressure deformities of the tibia and fibula. The trabecular structure is unusually coarse because of widening of the diploic spaces. Note the presence of phleboliths in the surrounding soft tissue tumor.

HEMANGIOMATOSIS

Multiple hemangiomas may develop independently throughout the body. Two of the patients examined showed diffuse involvement of the arm and forearm. Four displayed extensive disease of the thigh and leg. One revealed widespread hemangiomatosis of the toes, while another presented multiple soft tissue hemangiomas in both hands and both legs. The soft tissue changes were often accompanied by definite enlargement of the neighboring skeletal parts (Fig. 14, *A* and *B*).

One of the more striking examples of hemangiomatosis was a case of a sixty-five year old female who had had numerous blood vessel tumors since the age of three.

FIG. 14. Soft tissue hemangiomas producing secondary changes in bones. (*A*) Widening of the phalanges of the left foot. (*B*) The normal right foot is shown for comparison.

Many of these were removed when the patient was fourteen years old and others were excised ten years later. The lesions subsequently recurred and increased in size so that they were visible over the entire body with the exception of the right upper extremity, the lower half of the left leg and the left foot. In the neck and abdominal wall in particular, numerous subcutaneous, soft, bluish-red, nonpulsating vascular lesions were encountered.

Roentgen examination showed that both lower extremities were diffusely involved by soft tissue hemangiomas. The large nodular soft tissue masses could be seen projecting from the skin surface and similar nodules could be detected in the deeper soft parts as well. There were many phleboliths. Pronounced changes were noted in the feet; there was disappearance of the phalanges of a number of toes and the metatarsals were attenuated and spindle shaped (Fig. 15 *B*). There was cortical thickening in many of the long bones with peculiar wavy irregularities in marginal outline. Ill-defined bony overgrowths projected from the shaft in a perpendicular fashion and there seemed to be actual sequestration of bone in some areas. Somewhat similar, although less severe, changes were noted in some of the bones of the upper extremities. Many of the phalanges of the hands showed fairly typical angiomatous changes without accompanying soft tissue hemangioma in the immediate vicinity (Fig. 15 *A*). Although the patient is currently under treatment, the prognosis is poor due to the presence of chronic nephritis and hypertension.

A similar case was reported by Bonse.[9] His patient was a seventeen year old girl who showed numerous soft tissue tumors in the region of the right supraclavicular fossa and over the right hemithorax, right arm and back.

Fig. 15. Hemangiomatosis. (*A*) Multiple, fusiform, soft tissue hemangiomas of the left hand with cyst-like expansion of the phalanges and pressure deformities on the phalanges and metacarpals. (*B*) Nodular soft tissue tumors of the feet with a disappearance of the phalanges of four toes. Note the attenuated and spindle-shaped metatarsals. There were similar, widespread changes noted throughout the body.

SUMMARY

1. The spine and skull are the most frequent sites for the occurrence of primary hemangioma in bone and the roentgen features are usually characteristic. The "corduroy cloth" appearance prevails in the spine and the fine "honeycomb" pattern in the skull.

2. In the flat bones, the "sunburst" and "honeycomb" patterns are fairly regularly encountered, but the roentgen diagnosis is not always easy.

3. In other skeletal sites, it is not unusual to see a "soap-bubble" appearance and this is often combined with a "honeycomb" pattern.

4. In some instances, particularly in the ribs and clavicle, a hemangioma of bone may present findings closely resembling a malignant bone tumor such as an osteogenic sarcoma.

5. Soft tissue hemangioma as well as hemangiomatosis may involve the bone secondarily causing local deformities, enlargement, cortical thinning, and altered texture.

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RESPONSE OF FILM-SCREEN COMBINATIONS TO VARIATION IN ROENTGEN-RAY KVP.

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THERE has been increasing interest in the use of higher and higher kilovoltages for diagnostic roentgenography.

Over a long period of time and in the course of other work, we have gathered data on the response of different film-screen combinations to variations in roentgen-ray kilovoltages. This information is presented, along with the general procedure which was employed, in the hope that the results may be of practical interest to the radiologist in his choice of film-screen combinations and kvp. It is, of course, to be understood that this work ignores completely such problems as contrast and detail, which affect the reading of roentgenograms.

GENERAL PROCEDURE

In all cases a determination was made of the relative mas. required to produce a diffuse film density of unity.

Data for kilovoltages below 140 kvp. were obtained using both self-rectified and constant potential roentgen-ray equipment and a water phantom. A 280 kvp. Vanguard roentgen-ray unit was employed with a water phantom to obtain data for kilovoltages above 100 kvp. An overlap was thus provided for verification of the validity of our procedure and results at the higher kilovoltages.

Incident exposure dose measurements were made using standard self-reading pocket dosimeters, the energy and dose-rate response of which were well within the limits of these experimental conditions. The measurements were sufficiently well replicated to provide good confidence limits and excellent reproducibility was obtained over a period of years and a variety of conditions.

RESULTS AND OBSERVATIONS

The commercial film-screen combinations were examined for their response to the different peak kilovoltages. Representative results of the measurements for the most common combinations are shown in Figures 1 and 3. The relation of incident air dose rate to peak potential given in Figure 2 was used to develop the relation in Figure 3. Note the reverse trend beyond 200 kvp. It should be remembered that the skin dose, which includes backscatter, is also a function of kvp. A curve of the skin dose will have its minimum shifted slightly towards a higher kvp.

Table 1 presents the relative mas. required to produce the same film density at 180 kvp. for different commercial film-screen combinations. This kvp. was chosen

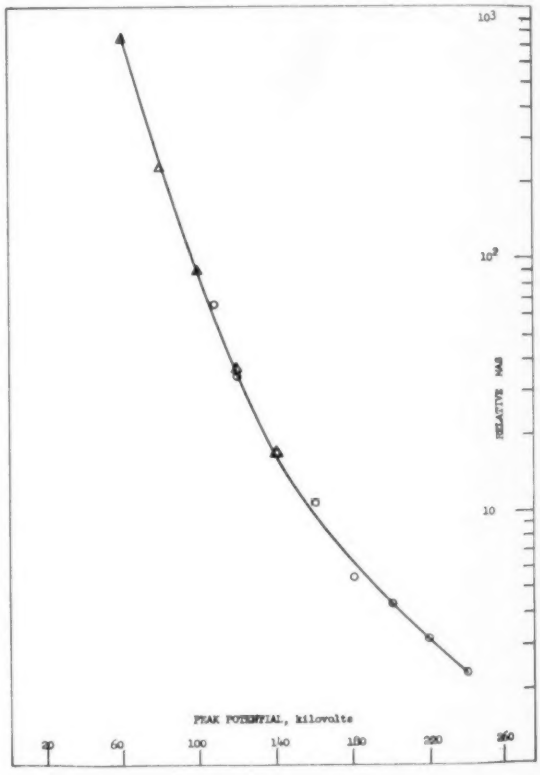

FIG. 1. Peak potential *vs.* relative mas. required to produce a diffuse density of 1.0 on standard film-screen combinations. ⊙—high potential roentgen-ray unit; △—low potential roentgen-ray unit.

Table I

RELATIVE mas. AT 180 kvp. FOR SOME FILM-SCREEN COMBINATIONS

Film	Screen	mas.
Kodak Blue Brand	du Pont Par Speed	6
Kodak Blue Brand	Ilford Standard	6
Kodak Blue Brand	Radelin General Purpose	6
Kodak Blue Brand	Ilford High Definition	13.5
Kodak Blue Brand	Radelin High Speed	2.5
Kodak Blue Brand	Radelin High Resolution	19
Kodak Blue Brand	Radelin Ultra Detail	22
Ansco High Speed	du Pont Par Speed	5
du Pont 508	du Pont Par Speed	7
Ilford Red Seal	du Pont Par Speed	6
Kodak Boyal Blue	du Pont Par Speed	5

merely because this is probably as high a kilovoltage as anyone is likely to use. The relationship with kvp. for all combinations

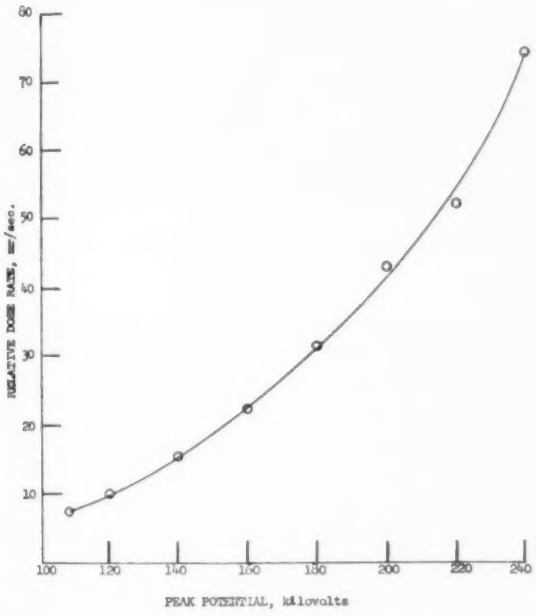

Fig. 2. Relative air dose rate *vs.* peak potential.

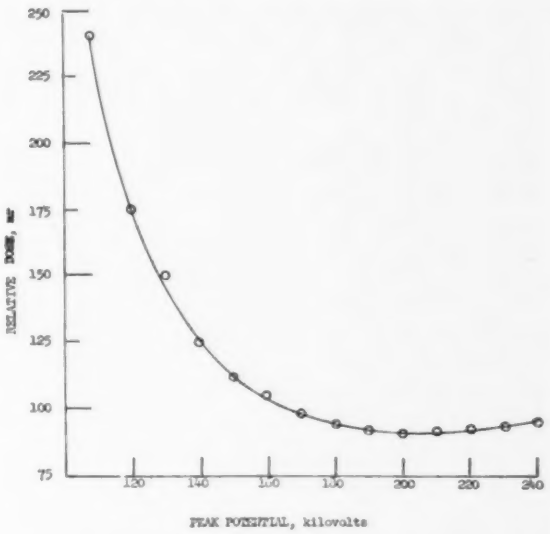

Fig. 3. Relative incident air dose *vs.* peak potential to produce a diffuse density of 1.0 on standard film-screen combinations.

except the Ilford H. V. screen is parallel to that of the common combinations shown in Figure 1. Our observations indicate that there is usually a relatively greater dose reduction obtained from the combination of a "high speed" screen with a standard film than from the converse, *i.e.*, a standard screen and a "high speed" film. This is in substantial agreement with data reported in 1948 by Van Allen.[1]

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We are indebted to Mr. Frank Dreisinger, Director of Medical X-Ray Education, for pointing up the medical interest in this problem.

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AN IMPROVED PATIENT HANDLING DEVICE*

By HUBERT S. SEAR, M.D., *and* FREDERICK J. DORAN
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HOSPITAL patients often are not only unable to stand, but even unable to move from a litter to a roentgenographic table. There is a need, therefore, for a device to assist technicians in moving these patients. Such a device should offer the following features: (1) for the patient it should be safe and should necessitate practically no exertion; (2) for the nurses and technicians who use the device, it should require the exertion of so little physical strength that it is easy for even an unassisted woman to use; and (3) for the hospital which pays for the device, it should be inexpensive and capable of being made in the maintenance shop. A device meeting these specifications has been developed. It was found, incidentally, that it allowed special roentgenographic projections not otherwise easily obtainable.

Figure 1 is a photograph of the device when not in use. A Hoyer lift,* which consists of a boom elevated by a hand operated hydraulic cylinder mounted on casters, was modified. The U-shaped base was widened in the hospital maintenance shop so that it would straddle the pedestal of the x-ray machines. A horizontal plate was then welded to the end of the boom. Chains with hooks at each end connected this plate with the U-shaped cradling device shown. The arms are about 19½ inches wide and 10½ inches apart.

FIG. 1. The appearance of the device when not in use.

Figure 2 shows two rods inserted in a canvas stretcher and engaged by the lift. A patient is seen suspended in the lift and positioned for a lateral chest roentgenogram using the table Bucky diaphragm. The suspension arrangement shown prevents the patient from swinging and reduces the need to change his position in order to balance the stretcher in the horizontal position.

The canvas stretcher is the key feature of this device. It is obvious that, once the patient is on the canvas, the lift can be easily engaged and the patient can be moved anywhere in the hospital without further effort. Since the canvas is radiolucent, roentgenography and fluoroscopy can be done through it without interference. The long bars in the stretcher lie to the sides, well out of the roentgen-ray field. Initially, the canvas can be worked under a patient by rolling him from side to side

* Manufactured by Ted Hoyer and Company, Oshkosh, Wisconsin.

* From the Department of Radiology, Lemuel Shattuck Hospital, Commonwealth of Massachusetts, Department of Public Health, Boston, Massachusetts.

in the same manner that the sheets are changed; or, alternatively, the orderly who is to move a litter patient may take a canvas stretcher with him and spread it over the litter before the patient is placed on it.

When this device was used in the X-Ray Department, the necessity for the orderly to make a judgment about the ability of a patient to stand or move by himself was eliminated by having every patient who needed a litter in order to get to the Department brought on a canvas stretcher. The decision to use the lift was then made in the Department.

A number of these canvas stretchers were available since they were easily made by the hospital sewing room. Soiling of stretchers due to accidents (as, for instance, those terminating barium enemas) was not a problem as they were easily laundered in the hospital.

It should be noted (Fig. 3) that, when the patient is lifted, there is considerable sagging in the canvas stretcher. This has three functions: (1) it prevents disoriented patients from rolling over the side; safety is further augmented, incidentally, by the position of the cradling device, which prevents the patient from sitting up; (2) when placed on an x-ray table, the excess material ensures that the rods fall over the sides and well outside the field to be roentgenographed; and (3) lateral roentgenograms can be obtained by placing the patient, suspended in the lift, against a wall-mounted film holder or against the table which has been rotated into the vertical position (Fig. 2). Excellent visualization of pleural fluid can be obtained in this way without effort by the patient.

FIG. 3. Slack, built into the canvas stretcher, allows decubitus roentgenograms to be obtained without effort and gives the patient a sense of security.

FIG. 2. The device holding a patient for a lateral roentgenogram of the chest, using the table Bucky diaphragm.

Several other applications of this device have been worked out in our hospital. Bed patients can be weighed by inserting a spring scale between the boom and the center of the cradling device. Paraplegics, supported on a tighter stretcher, may be safely bathed in an ordinary bath tub.

Also, there appear to be obvious applications in other departments. The transfer of anesthetized patients from an operating table after a delicate operation is sometimes roughly performed. Similar handling

of patients delivered from the accident ward (with fractures, for example) does not help their treatment, yet up to now was unavoidable. This device can make patient handling in these circumstances safer for the patients and effortless for the personnel.

The device has been in service for over a year and, by performing the functions mentioned above, has gained considerable acceptance by patients, physicians, nurses and technicians.

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Lawrence Reynolds
1889-1961

EDITORIALS

Lawrence Reynolds and The American Roentgen Ray Society

With the death of Lawrence Reynolds, the American Roentgen Ray Society in particular and medical journalism in general have suffered a loss which will be felt for many years to come. Although Dr. Reynolds was a vigorous member and eminent President of this Society, as well as of several others, he will be best remembered for his immeasurable contribution as Editor of the American Journal of Roentgenology, Radium Therapy & Nuclear Medicine, the official organ of the American Roentgen Ray Society and the American Radium Society. Dr. Reynolds was first associated with the Journal as an Assistant Editor in 1928. In 1930 he became the editor and, until his death, he held that position continuously with the exception of the years 1947–1949 when he was President-Elect and President of the American Roentgen Ray Society. I doubt that anyone can really appreciate the amount of time and effort that Dr. Reynolds so selflessly gave to the Society in those thirty years, but the results are obvious to even the most casual reader of the Journal. Without exaggeration or fear of contradiction, one can truthfully say that the envious position which the Journal now occupies in the field of medical publications is, in a very large measure, due to the untiring, skillful and tactful editorial managing by Dr. Reynolds.

It can be stated that Dr. Reynolds was a "natural" for the position of Editor, for not only did he have the necessary intellectual interests and abilities but he also possessed limitless patience and great tact combined with a remarkable administrative capacity and a wonderfully warm personality.

Dr. Reynolds' avocations, too, were centered around literary activities and during his lifetime he assembled a valuable collection of rare and first edition medical books. A number of libraries throughout the country made use of Dr. Reynolds' wide knowledge and experience in activities of this sort and for years he served on the library governing boards of such institutions as Yale University, University of Michigan, Wayne County Medical Library Commission and the City of Detroit Library Commission.

The American Roentgen Ray Society knows full well that it can never adequately express its appreciation for Dr. Reynolds' years of distinguished and devoted service. Nevertheless, it does wish to record on this page in these few feeble words the position of great honor and affection he so rightly deserved and in which his memory will always be held.

John F. Roach, M.D.
Chairman of the Publication Committee

Resolutions of the Detroit Roentgen Ray and Radium Society

WHEREAS *the death of Doctor Lawrence Reynolds occurred on August 17, 1961 and*

WHEREAS *Doctor Reynolds was one of the earliest and most eminent members of this Society and*

WHEREAS *he brought renown to this Society by the excellence and multitude of his scientific productions and by his great ability as a teacher and as a practitioner of our profession and*

WHEREAS *his great energy and humanity and his concern for and excellent care of the afflicted served as an example for our membership and*

WHEREAS *he set a high standard for our practice and was at all times to us a teacher and friend, be it therefore*

RESOLVED *that his death be recorded in the minutes of this Society with a feeling of high regard for his memory and a deep sense of loss to all members of the Society, and be it further*

RESOLVED *that a copy of this memorial be submitted to the* AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY & NUCLEAR MEDICINE *and to* Radiology.

Lawrence Reynolds and Harper Hospital

WHEN Lawrence Reynolds was graduated from the Ozark, Alabama, high school in 1907, his teacher, Mary Newton, presented him with two volumes of Oliver Wendell Holmes' "The Autocrat of the Breakfast Table." Mary Newton's inscription read: "Lawrence: Acquire the genial humor, the ready wit, and the broad culture of the recondite old aristocrat."

That he succeeded in fulfilling Miss Newton's fondest hopes for him, all of his associates at Harper Hospital can affirm. It has been my privilege to serve as President of the Board of Trustees during the entire time of Dr. Reynolds' association with Harper Hospital and to observe his influence in our institution.

This young doctor, dedicated to a young specialty, came to us shortly after the conclusion of World War I. He lived and worked as an influential member of our "Family" from that time until his death. He was devoted to his patients, his profession, and to the welfare of this institution, and his devotion was enriched by "genial humor," "ready wit," and "broad culture."

These qualities gave him insight into problems frequently onerous to the practicing physician, problems like the practical ones of institutional organization which face a Board of Trustees. He knew, too, that the physician's ethics and ideals frequently impose conditions which perplex even the most informed laymen, like members of a Board of Trustees. He knew that each body needed interpretation to the other so that they could work together to fill their common purpose. And he was uniquely fitted, largely through practice of his old teacher's advice, to fill the function of interpreter. Dr. Reynolds, throughout his career at Harper Hospital, had an unusual comprehension of the legitimate interests of both. As Chief of our Department of Radiology, as well as in his later position as Chief of the Medical Staff, he worked ceaselessly for that ideal balance which inevitably results in institutional progress.

Illness was the only reason for his ever missing a meeting of the Board of Trustees during his tenure as Chief of the Medical Staff—and happily, there were very few such occasions. Ours was a long and pleasant association. He gave generously of his personal time and abilities to ensure that our mission could be effective in our community.

It was not only in his professional life that Dr. Reynolds was a man of "broad culture." His name could always be found in the list of sponsors of any worthwhile community endeavor. This was not a perfunctory listing, for where his name appeared, he was active, and his active participation served our hospital well. One always knew that Dr. Reynolds' interpretations of need, and recommended actions on behalf of the Medical Staff he represented, were based upon the ultimate welfare of the patient, and that he had carefully considered this in the light of his knowledge of our community, as well as in the light of the profession he served so well and faithfully. We are grateful that he was with us for most of his long and fruitful career, and we shall sorely miss him.

RICHARD H. WEBBER, *Chairman, Board of Trustees, Harper Hospital*

Lawrence Reynolds and Wayne State University

Doctor of Laws

June 14, 1956

LAWRENCE REYNOLDS,

a native of Alabama; a graduate of the University of Alabama in 1912, and of the Medical School of the Johns Hopkins University in 1916; he has carried on the private practice of roentgenology in Detroit since 1922, and for eleven years has been Professor of Radiology in the Wayne University Medical School. Since 1948 he has served as Chief of the Department of Radiology, and as Chief of Staff and Chairman of the Executive Committee of the Harper Hospital, Detroit.

Honored by his alma mater with the degree Doctor of Laws, and by radiological societies in Germany, Italy, and Colombia with honorary memberships, he has edited the *American Journal of Roentgenology*, *Radium Therapy*, and *Nuclear Medicine* since 1936.

Through the years he has brought together one of the world's great private collections of medical texts and treatises. Noted for his knowledge of library acquisitions, he is currently an Honorary Trustee of the Yale Medical Library Associates, a Member of the Committee of Management of the Clements Library of the University of Michigan, Chairman of the Medical Library Commission of the Wayne County Medical Society, a Member of the Grolier Club of New York City, and of the Harvey Cushing Society. As President of the Detroit Public Library Commission, he has brought to local problems the breadth of view and deep wisdom of his national experience.

Wayne University is proud to recognize the work of one of this country's greatest radiologists. His lifetime devotion to scholarly work as editor and bibliophile, his own research and his encouragement and training of young scientists, and his direction of the staff of a great hospital have helped to shape the whole course of modern radiological medicine.

IN THE passing of Lawrence Reynolds, the cause of scholarship and intellectual excellence lost a firm friend. His entire medical career was one in which the teacher, investigator, bibliophile and practitioner of the skills of radiology were artfully blended. His service to medical education was a long and varied one. When he returned from overseas service in World War I, he took a post as Instructor in Roentgenology at the Johns Hopkins University Medical School. This appointment so soon after graduation was indeed a rare thing in academic medicine in the period before 1930. The following year he went to Boston to Harvard and the Peter Bent Brigham. He remained in Boston as roentgenologist at the Brigham and Instructor in the Harvard Medical School.

In 1922 Dr. Reynolds came to Detroit and entered the private practice of roentgenology. However, this activity gave him only partial satisfaction. The world of books and the world of learning which had captured his attention as a youngster would not be denied. Along with his busy professional life, he began to collect those volumes which are now the care of the Reyn-

olds Library in the University of Alabama. His knowledge of medical history grew with the library. His fine discrimination and his desire to achieve perfection made it possible for him to assemble a collection which contains the finest copies extant of many of its items.

In 1945 Dr. Reynolds was appointed Professor of Radiology and Chairman of the Department of Radiology in Wayne State University. He served the University well for seven years. In this time he brought to life a department which had suffered through the agony of World War II as well as chronic budgetary inanition. When this had been done, he urged the prompt appointment of his successor and resigned as Chairman of the Department. He continued to serve the University as Clinical Professor of Radiology until his seventieth year when he became Professor Emeritus. He brought great distinction to the College of Medicine and faithfully attended to his duties as teacher, especially in the areas of graduate instruction and resident education.

In recognition of his great attainments in the world of books, his service to the Public Library of Detroit, the Yale Medical Library, Clements Library of the University of Michigan and to Wayne State University, Dr. Lawrence Reynolds was granted the degree of Doctor of Laws in 1956. This was but one of many honors which came to him the last fifteen years of his life. He deserved them all. His colleagues will long remember him with respect and affection.

GORDON H. SCOTT, *Vice President for Medical College Development and Dean, College of Medicine, Wayne State University*

Lawrence Reynolds as a Teacher

"I WILL share my precepts, oral lessons, and any knowledge with my sons, with those of my master, and with pupils bound by a promise and an oath in accordance with medical law." A covenant injected into the medical conscience of all of us by Hippocrates, unrequited in the activities of many, but accepted each day by Lawrence Reynolds, M.D. As a proponent of the swift accurate interpretation of the roentgen shadow, he certainly taught all by example.

And yet, teaching was more to him than this.

At some time during his long career he became imbued with the importance of diffusing medical learning. Perhaps this developed as he read to his blind physician father in rural Alabama. A frequent text was Sir William Osler's "Principles and Practice of Medicine." The latter's views on teaching were strikingly similar to his own. "Replace the Chinese with the Greek spirit, enable him (the student) to seek knowledge for itself . . . the pupil and the teacher working together on the same lines, only one a little ahead of the other." Dr. Reynolds approached education as though he felt that teaching was the perpetual Messiah of medicine; the constant connection between the established and the aspiring, the bond between accepted fact and better ideas. He cultivated the ability to instill an intellectual respect within the student without pre-empting questions or rebuttal. He knew how to sustain the interest of his residents, plant the seeds of original investigation, or at times to spoon feed when this was the most expedient kind of informative nutrition. His greatest talent was his ability to prevent his mere presence or renown to persuade or unduly influence his residents or students. He chose to counsel, but not to overawe.

Training in radiology began at Harper Hospital in 1920. Somewhat later a formal residency program was inaugurated. For many years punctually at 7:30 A.M., residents and, subsequently, students from the Wayne State College of Medicine clustered about Dr. Reynolds in the reading room. These sessions usually lasted for two or three hours. He disdained the formal lecture, preferring the spontaneity of the day's cases for his teaching purposes. Protocol was limited to the painless introduction of the neophyte into the discussion with "Would you continue to employ this man with a chest that looks like this?" He was the faculty and all of radiology was his curriculum. His film descriptions were always punctuated with shrewd clinical insights or embellished by the recall of a conversation with Drs. Harvey Cushing, Henry Christian or some other titan. The mornings were further enriched by his lore of the history of radiology and colorful sketches of its pioneers. Retelling one of Sir William Osler's stories about himself, he pointed out the importance of a circumspect differential diagnosis. Shortly after Dr. Osler came to Baltimore he was asked to see an elderly and important gentleman. Examination

revealed a large pelvic tumor, and a diagnosis of inoperable sarcoma of the pelvis was promptly made. As gently as possible Dr. Osler explained the condition to the patient's wife and advised that a surgeon see the case. The same day the surgeon came and removed the tumor with a catheter. Similar pointed, often humorous stories usually adjourned our meetings.

He schooled his residents in good radiologic manners and practicality. His maxims were simple. Courtesy should be the trademark of the radiology department. Service is the prime consideration extended to the attending physician. Examinations should be conducted quickly with initial accuracy. We were always cautioned to examine and talk to the patient so as to be able to extract the most from the roentgenograms. He once said that a radiologic report should read like the Egyptian Rosetta Stone: meaningful to all who might have occasion to peruse it.

As a Charter Diplomate of the American Board of Radiology he was well acquainted with the scope and aims of its certifying examinations. He had a recurring penchant for keynoting the diagnostic entities and procedures likely to be emphasized from time to time. Grooming such as this helped to lessen the incubus of intensive study and provided the tangible tools for shaping a profitable plan of preparation.

A certain filial charm fitted him well for his role as Professor of Radiology at The Wayne State University College of Medicine. The young men, many of whom affiliated with the program largely because of Dr. Reynolds, rightfully came to expect his facile solutions to their most perplexing medical and personal problems. He was adviser, confidant and intercessor to many. In those interns, students and residents merely passing through the x-ray department, he tried to create an appreciation for the proper place of radiology in the spectrum of medical specialties and for the role of the radiologist in the care of the patient.

The literature abounds with his medical papers and editorials, and these, too, serve to convey knowledge. They cannot supplant the daily *vis-à-vis* contact we had with this superb radiologist. He was available. He was approachable. His time was not of the essence. He never spoke from a professorial whirlwind. He passed the baton of knowledge with the grace of an Olympian.

In journeyman fashion our local newspapers concluded, "Doctor Reynolds leaves no immediate survivors." More correctly, he left none who survives him only immediately. Some of us are his disciples; some his ambassadors, a very few may become his peers. We all have a legacy in his encouragement to try to learn something from everyone. Through his teachings we link him with tomorrow. In the words of Henry Adams, "A teacher affects eternity; he can never tell where his influence stops."

JAMES J. SNIDER, M.D., *Former Medical Student, Wayne College of Medicine and Assistant Radiologist, Harper Hospital*

The Lawrence Reynolds Library*

On Sunday afternoon, February the Second 1958, The Lawrence Reynolds Library was dedicated by impressive ceremonies at the University of Alabama Medical Center in Birmingham. This Library, which contains one of the world's finest and most valuable private medical collections, was donated to his Alma Mater and his native state by Dr. Lawrence Reynolds, Editor of this Journal, who spent a lifetime acquiring it.

This rare collection is housed in a special wing which the University has built as an extension to its new Medical Center Library. It consists of nearly 6,000 individual pieces of original manuscripts, autographed letters and old medical books, some of them dating back to the fifteenth and sixteenth centuries, representing samples of highest achievement in Renaissance printing.

The section of original manuscripts includes, among others, the fourteenth century manuscripts of Arnoldus de Villa Nova entitled *Regimen Sanitatis ad regem Aragonum* and John of Toledo's *Dietarium*; the original manuscript of the article on *The Stereoscope and Stereograph*, contributed to the *Atlantic Monthly* in June, 1859 by Oliver Wendell Holmes; and, of the more recent ones, the address on *The Discovery of Radium* by Mme Marie Curie (Sklodowska) delivered on May 14, 1921 at Vassar College, Poughkeepsie, New York, with an autographed photograph.

The letter section contains all the correspondence of Louis Pasteur with the Mayor of the village on silk worm disease, consisting of 20 holographic letters of Pasteur (1866–1889), 8 letters in the hand of Mme Pasteur, and 2 unpublished photographs; the letter of Louis Pasteur to Monsieur Marchand, dated August 12, 1881, on rabies; and other items from the pen of this giant of science who revolutionized both industry and medicine by applying laboratory knowledge to fight disease. There are also signed letters with typed transcripts by Sir William Osler to his "good friend Ned Milburn" (1865–1919), and two original letters from Wilhelm Conrad Röntgen and Frau Wilhelm Conrad Röntgen to Louis Grauel; and others.

Of the large section of medical books of the sixteenth and seventeenth centuries, three groups deserve special mention. It is now generally agreed that the foundations of modern medicine were laid down in this period by three epoch-making contributions. In 1543 the first complete text book on human anatomy was published by Andreas Vesalius entitled *De humani corporis fabrica librorum epitome*, Johannes Oporinus, Basel. This book, as well as Vesalius' other contributions, was illustrated with magnificent drawings by van Calcar, a pupil of Titian. The Library contains not only Vesalius' textbook in its first edition but also other works of the truly great anatomist, several later editions of his works and a group of books on the works of his

* Reprinted from: Am. J. Roentgenol., Rad. Therapy & Nuclear Med., April, 1958, *79*, 726–727.

teacher, Sylvius, who from a friend turned into a bitter foe, of Fallopius and Eustachius, his successors, and of other famed anatomists of the period, most of whom became "Vesalius converts." This study of the "fabric" of the human body based on pure research was soon followed by an interest based on similar ground in the functions of the human body. Among those who pursued such a course was Andreas Caesalpinus, who, in his *Peripateticarum Questionum Libri Quinque* published in Venice in 1571, began speaking of the "circulation" of the blood. His bold theory, however, was not supported by experimental proof and thus he had no influence on his contemporaries. Later in 1628, at the book fair in Frankfurt, a slender, poorly printed volume was displayed by William Harvey on the *Exercitatio anatomica de motu cordis et sanguinis animalibus*, presenting for the first time experimental proof that the heart acts as a pump causing circulation of the blood throughout the body. The books of Caesalpinus and Harvey and 12 other texts and facsimiles of the book of Harvey published in different countries form the nucleus of the second group of works, containing the contributions of Fabricius Hieronymus of Aquapendente, Harvey's teacher, on *De venarum ostiolis;* of Francis Bacon, one of Harvey's most notable patients, on *Instauratio magna* (*Novum organum*); Harvey's second great work *Exercitationes de Generatione Animalium* (1651) on embryology and many similarly rare items. The third group of collections of this period is highlighted by works of Ambroise Paré, the greatest surgeon of the sixteenth century. Paré based his practical surgery on the new concepts expounded in Vesalius' *De Fabrica* and, after being able to conquer the prejudices of the time, introduced sound methods for the treatment of a rather significant number of lesions. His influence on contemporaries and successors for the next two centuries is amply reflected by the many contributions comprising this group.

In 1600, William Gilbert published his epochal work *De magnete, magneticisque corporibus et de magno magnete tellur; physiologia noua, plurimis argumentis experimentis demonstrata*, establishing the basis for the formidable sciences of magnetism and electricity, which were to develop. The early progress was rather slow until the middle of the eighteenth century when Benjamin Franklin in his extensive *Experiments and Observations on Electricity Made at Philadelphia* defined "positive" and "negative" electricity and considered, among many other things, the possibility of curing palsy by electric shock. In 1786 Luigi Galvani discovered "animal electricity." This was followed in rapid succession by Alessandro Volta's construction of the first electric pile, as communicated in a letter to the Rt. Hon. Sir Joseph Banks (1800); by Hans Christian Oersted's *Experimenta circa effectum conflictus electrici in acum magneticum* (1820), establishing a link between electricity and magnetism; by André Marie Ampère's *Mémoire présenté à l'Académie Royale des Sciences sur l'action mutuelle entre deux courants électriques* (1820), describing the mutual effect of two electric currents; and by G. S.

Ohm's work in 1827, formulating the relationship between voltage, amperage and resistance. Then in 1837, Faraday and Henry discovered electromagnetic induction. These important works and many others in the chain of development of the two sciences form an eminent part of the Library and constitute a monumental introduction to the vast collection of more recent contributions in the field of radiology.

Among the more recent contributions are also many rare works from other branches of medicine, as exemplified by some 40 writings from the pen of Sir William Osler, the great physician of modern times; an almost complete set of Harveian Orations, beginning in 1865; an extensive compilation of Nobel Prize Papers; and others.

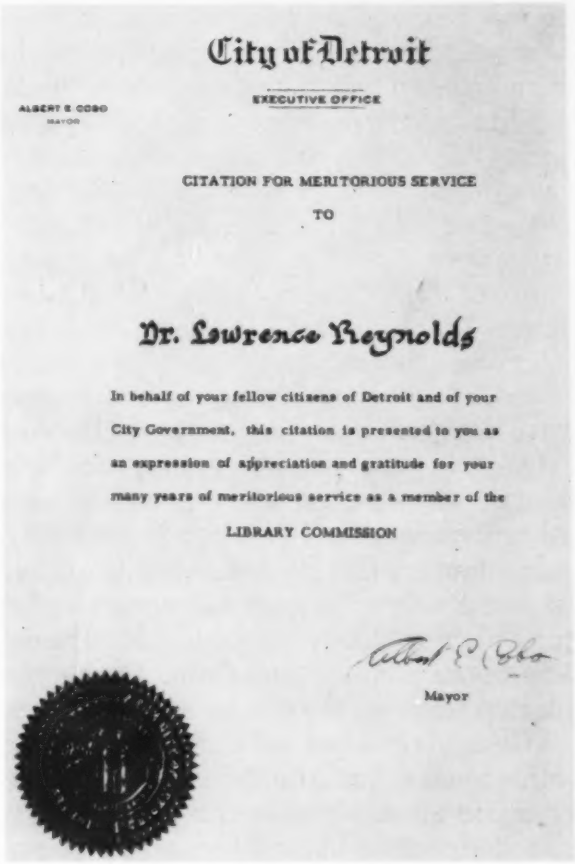
These are but brief heuristic glimpses into a magnificent Library. It is written on the dedication plaque that graces its entry: "Each time one of you reaps from the great minds of the past the desire for finer achievement in your profession and nobler development of your own character, The Reynolds Library will have been rededicated." In the ancient words of Terentianus Maurus spoken in 1286, *Pro captu lectoris habent sua fata libelli.*

TRAIAN LEUCUTIA

Lawrence Reynolds and the Detroit Public Library

In December of 1960, Dr. Lawrence Reynolds, for the third time, completed a term as President of the Detroit Library Commission. It is indicative of his thinking that the final paragraph of his last presidential report included this quotation:

"What may be emerging as a mark of our own generation is a widespread renewal of concern for the quality and intellectual aims of education."



City of Detroit

EXECUTIVE OFFICE

ALBERT E. COBO
MAYOR

CITATION FOR MERITORIOUS SERVICE

TO

Dr. Lawrence Reynolds

In behalf of your fellow citizens of Detroit and of your City Government, this citation is presented to you as an expression of appreciation and gratitude for your many years of meritorious service as a member of the

LIBRARY COMMISSION

Albert E. Cobo

Mayor

This statement, quoted from Jerome S. Bruner, appealed to Dr. Reynolds because for the seventeen years during which he served as a member of the Detroit Library Commission he consistently supported every effort to make this Library one which was distinguished for excellence in its book collections and in its service.

Dr. Reynolds was first appointed to the Detroit Library Commission in 1944, to fill an unexpired term, and was reappointed to six-year terms in 1949 and 1955. Thus he was just completing seventeen years of continuous service, more than any other current Library Commissioner, when his death occurred. The years spanned by his service were unusually important ones for the Library: the departmentalization of the Main Library was completed with the establishment of five new departments; ten new branch libraries were erected; a $500,000 service building was built to house the Library's extensive building maintenance shops and its large book bindery; and finally, after years of striving to have the beautiful Main Library enlarged, construction of a ten-million-dollar addition was started. It is regrettable that he didn't live to see the building occupied, which it is anticipated will be about a year from now.

Though he recognized the need for adequate buildings for the Library, his

interests were largely centered on books and their use. He revered fine books, and frequently visited the Rare Book Room; he respected the printed page of any book whose author had integrity and competence, and he had an active interest in trying to make library service available to adults and children, to scholars and beginners, to specialists and to those whose reading is broad in scope. It is interesting to note that while he was busily studying the library's books on small country libraries, with a view to helping his home town, Ozark, Alabama, develop a sound beginning for a community public library, he was also involved in providing a firmer base of support for the medical library in Detroit. The culmination of the latter is best covered by quoting from his 1949 report as President of the Detroit Library Commission:

"*The support, administration, and function of the Medical Science Library having become more and more confused through the years, it was finally decided by the Detroit Library Commission that the best interest of the people of the City of Detroit would be realized by putting its 51,000 volumes under the management of the Medical School of Wayne University. . . . By the accomplishment of this transfer, the sole responsibility for the care and maintenance of this Medical Science Library rests with the Medical School of Wayne University.*"

The instrument of transfer, however, at Dr. Reynolds' insistence, contained assurance that the Medical profession of the Detroit area should always have free and ready access to the Medical Science Library.

Quite apart from whatever his accomplishments were in his chosen profession, he was a person who quietly and unselfishly assumed more than his full share of obligations to society in general.

In manner, Dr. Reynolds has been described as courtly. An innate graciousness, a sensitive consideration for the feelings of others, and a pleasant, quiet dignity characterized him at all times. These fine qualities were as evident to the building guards and the elevator operator as they were to the administrators and the Commissioners of the Library.

Within days after his death, a very beautiful tribute from a non-medical professional acquaintance in New York was received. Two paragraphs of that letter are quoted here, as they pay tribute to him as an outstanding person who distinguished himself in service given beyond his own profession.

"*He was a man of culture, intelligence, energy and breadth of vision, and therefore highly qualified as a member of the Library Commission.*

"*This physician typified to me what a fully developed human being should be, and it is comforting to realize that the human race from time to time can produce such an outstanding scholar and citizen.*"

RALPH A. ULVELING, *Director, Detroit Public Library*

The Heritage of Lawrence Reynolds

A MAN'S heritage may in part determine his course through life. Many, imbued with the finest of inheritance, fail to appreciate its value, to utilize it, enhance it and to pass it on with enrichment to those who follow. Few men have succeeded so admirably in nurturing and embellishing an already rich heritage as did Dr. Lawrence Reynolds.

Born in lower Dale County, Alabama, Dr. Reynolds was reared among gracious, hospitable people in the post reconstruction period of our nation's history. That this portion of his heritage may have been changed is related in an anecdote known to his family and pertains to the emigration from the State following the Civil War. Two such movements occurred,[1] the first culminating in 1869 and the second in 1873–1874 with most of the emigrants going to Texas, while others went to Brazil, Mexico, the North, Tennessee and Georgia. It was estimated at that time that the State lost more of its population by this emigration than by the effects of the war itself. Dr. Reynolds' grandfather, it is said, may have been prompted to participate in this emigration but was induced to stay in his beloved Alabama to care for the local populace.

The Reynolds family[2] is descendant from English and Welsh immigrants to this country in the early seventeenth century who settled on the Rapidan River, Isle of Wight County, Virginia in about the year 1637. An ancestor, George Reynolds, was born in 1555 in County Kent, England and gave issue to some children who settled in Virginia while others came to the New England states. Two well known descendants of George Reynolds were Samuel Langhorne Clemens, known to all the world as Mark Twain, and Nancy Witcher Langhorne who later became Lady Astor. Dr. Reynolds shared his family heritage with these distant cousins along with many distinguished military officers and other persons of eminence.

The Reynolds coat of arms, the proud possession of a great family, had its beginnings in the days of antiquity. The family's ancestry is traced to Norman origins claiming descent from Reynolds, son of Greyferth—The

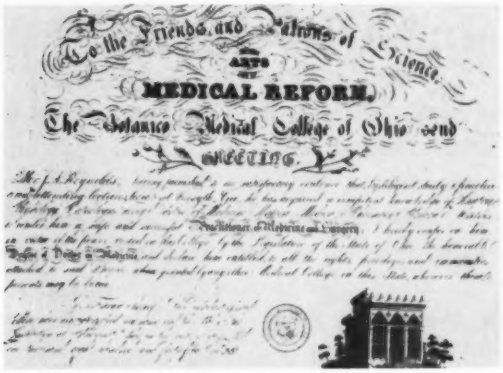
[1] Civil War and Reconstruction in Alabama. Walter L. Fleming, Columbia University Press, New York, 1949.

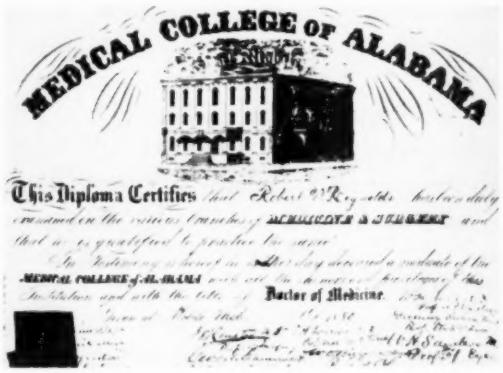
[2] Reynolds Family—1530–1959. The Goetz Company, Washington, D. C., 1959.

Dane—King of Northumberland. Greyferth and his two sons were driven from England about 944 A.D. and took shelter in Normandy. Fitzreynolds (meaning son of), one of the sons of Reynolds, returned to England with William the Conqueror.[2]

Dr. John Adams Reynolds, Lawrence's paternal grandfather, was born in 1819 in North Carolina and moved to Alabama at the age of seventeen, later practicing medicine there for a period of forty-seven years. His medical diploma from a school in Cincinnati is dated February 23, 1845. He died in 1891, two years after Lawrence's birth and, therefore, the grandfather's heritage of medicine was handed down through his son, Dr. Robert Davis Reynolds, born in Alabama in 1845, who had his preliminary medical education in Mobile in 1872, just seven years after the close of the Civil War.

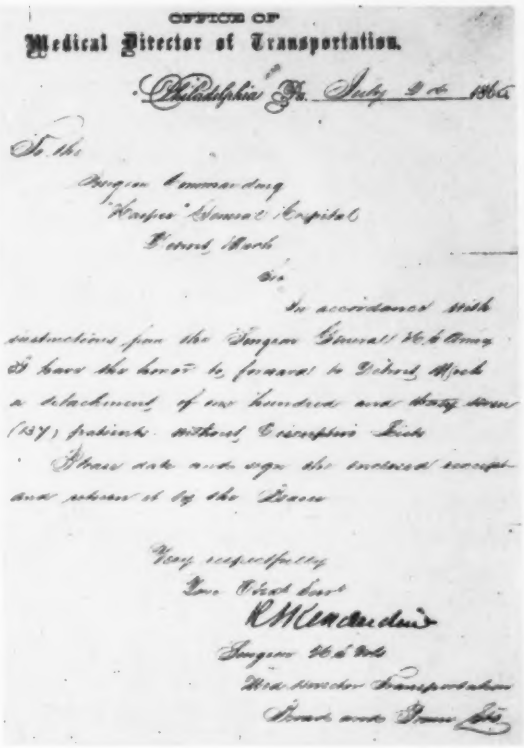
"Dr. Bob" served as a First Sergeant, Company F, 6th Alabama Cavalry, having enlisted in April, 1863 at the age of sixteen in T. A. Abercrombie's Division. After his return from the war, Dr. Bob went to the medical school in Mobile and in 1872–1873 practiced with his father at Skipperville in Dale County until the fall of 1880 when he returned to the Mobile Medical School and graduated with the degree of M.D. He then practiced in Skipperville for nineteen years, subsequently moving to Ozark, Alabama. Dr. Robert Davis Reynolds was blind throughout a large part of his professional life. Thus





young Larry became his father's eyes as he went about his rounds. Exposure to the practice of medicine in this somber and impressive way reminds us of Sir William Osler's "The Student Life," in which he states "Amid an extreme heritage of sorrow and suffering our work is laid." Undoubtedly this is the medical heritage which induced Lawrence to become a physician, one who never lost the warmth that is so necessary in the proper care of the sick. Lawrence Reynolds went to the Johns Hopkins Medical School in 1912, subsequently entered World War I, returned to Johns Hopkins, and later became associated with Peter Bent Brigham Hospital and Harvard University in Boston. In 1922 he came to Harper Hospital in Detroit.

Harper Hospital had its birth during the War between the States and it was through Dr. Reynolds' interest in this conflict that a copy of an original order from the Surgeon-General of the United States Army, dated July 26, 1865, to convey a detachment of 137 soldier-patients to the "Harper" U.S.A. General Hospital was procured and presented to the historical collection of the Harper Library.


OFFICE OF
Medical Director of Transportation.

Philadelphia Pa. July 26th 1865

To the
Surgeon Commanding
Harper U.S. General Hospital
Detroit Mich

Sir

In accordance with instructions from the Surgeon General U.S. Army I have the honor to forward to Detroit Mich a detachment of one hundred and thirty seven (137) patients without Descriptive Lists

You will take and sign the enclosed receipt and return it by the Bearer

Very respectfully
Your Obed't Serv't
R M Clendenin
Surgeon U.S.A.
Med. Director Transportation
Board and Transportation

Originally Harper Hospital was a series of eleven frame buildings facing Woodward Avenue. At the end of the war, a local Board of Trustees assumed control of the hospital with the provision that Civil War soldiers would continue to be cared for.

Later, in 1863, Harper Hospital became the site of the Detroit Medical College, the first medical school in the City, utilizing two of the eleven buildings. In January, 1884 the Children's Free Hospital Association was formed and Harper Hospital served as the first children's hospital until December, 1886 when the facilities at Harper were outgrown and a new location was obtained on West Fort Street. An early lying-in facility, later resulting in the formation of Woman's Hospital, was also initiated at Harper Hospital. Still other institutions, such as St. Luke's Hospital and Church Home in 1867, had their beginning at Harper Hospital.

Thus Dr. Reynolds became associated with a hospital which later was to achieve eminence among American hospitals but had its roots in the Civil War. The writer was privileged to be along with Dr. Reynolds, a charter member of the Harper Hospital Civil War Study Club, organized on February 12, 1958.

Dr. Reynolds' ability to enrich his heritage is further manifested by his affectionate relationship with the scholarly Dr. Hickey, whom he first met in the European Theater of World War I and who was the Founder and First Editor of this JOURNAL which Dr. Reynolds then subsequently edited for a period of over three decades.

From this great inheritance of family, the South, the medical institutions

with which he was early connected, and his associates, Dr. Reynolds brought to his professional life charm, warmth and enthusiasm which have been an inspiration to many. Sir William Osler has said, "No bubble is so iridescent or floats longer than that blown by the successful teacher." Dr. Reynolds has blown many such bubbles in his illustrious career. To those of us privileged to have been associated with him the words from Psalms 16, Verse 6 are particularly pertinent, "The lines are fallen unto me in pleasant places; yea, I have a goodly heritage."

KENNETH L. KRABBENHOFT

Lawrence Reynolds—An Homage

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE in its October, 1961 issue regretfully announced the death of its distinguished Editor, Dr. Lawrence Reynolds, which occurred on the evening of August 17, 1961 in Detroit, Michigan. The end came suddenly, with quiet grace and dignity, as he would have wished it to be—befitting his gentle nature and noble character.

Lawrence Reynolds was born February 11, 1889 in Skipperville, a charming community near Ozark, Alabama. He was the eighth of nine children of Dr. Robert Davis Reynolds, Sr., a practicing physician of that community, and of Mary Frances Card. Two of his brothers, John Leonard Reynolds and Robert Davis Reynolds, Jr., as well as his grandfather, John Adams Reynolds, were also physicians. This excellent family background afforded him a wisely directed early education which undoubtedly carried the seeds of a brilliant medical future. After graduation from the Ozark High School in 1907, he attended the University of Alabama and in 1912 received the degree of Bachelor of Arts.

During these formative years of Lawrence Reynolds, the elder Reynolds gradually lost his eyesight and from the age of twelve the inseparable son became the "eyes" of his beloved parent, driving his horse and buggy on house calls, writing out his prescriptions and acting generally as his most trusted adviser. The cultured father was a great admirer of classical literature and was keenly interested in Sir William Osler and other contemporary medical giants, so young Lawrence did the thing that would give his father the most pleasure—he read to him night after night the texts that he was so fond of hearing. This beautiful expression of filial attachment imperceptibly ripened into an inquisitive appreciation and love of his progenitors' noble profession and the fall of 1912 found Lawrence Reynolds at the Johns Hopkins Medical School, eager to absorb knowledge from such famous men as William H. Welch, William S. Halsted, Joseph C. Bloodgood, Hugh Hampton Young, Howard A. Kelly and others.

In June, 1916, upon receiving his M.D. degree, he timidly approached Dr. Frederick H. Baetjer, Professor of Radiology of that renowned institution, and within a short time became his first resident, thereby starting on an unexplored trail which was to blaze for him so brightly later.

In May, 1917, restless, now radiologically indoctrinated, young Lawrence was ready to move again, this time overseas with the American Ambulance Service, to care for the suffering soldiers in the European Theater of War. In view of the fact that the Department of Radiology was under the administration of the Department of Surgery, it was necessary for him to obtain "clearance" from the Chief, Doctor Halsted. This was granted rather reluctantly, with the provision that within six months he would return to his old post.

In the meantime, World War I had reached the climax in its fury. Captain Lawrence Reynolds remained with the A. E. F. for two years. During this period it was his good fortune to be the recipient at the American Ambulance Hospital No. 1, later called the American Military Hospital No. 1, of the first Coolidge tube overseas which later was to revolutionize therapeutic radiology. This tube was presented for inspection by him to General Pershing on his visit to the hospital.

Another of his good fortunes, and undeniably the most fateful event in his life, was to meet, while in the European Theater of War, among the many Allied radiologic celebrities, our own Major Preston M. Hickey, who went to France with the American Expeditionary Forces early in 1917 as roentgenologist with the Harper Hospital Unit—one of the first to be sent overseas. Major Hickey was early detached from that duty and assigned to the larger duty of Consultant in Roentgenology, visiting the base areas throughout France as an adviser to those charged with administration of the x-ray work in the entire A. E. F. In this capacity, Major Hickey often visited the American Military Hospital No. 1 and always found Captain Reynolds hard at work, performing his exacting duties with faithful thoroughness, yet with a genial manner and complete equanimity. These comradely meetings under difficult circumstances later led to an enduring friendship and an extremely valuable collaboration between the distinguished Founder and the tridecennial Editor of this JOURNAL.

In May, 1919, Lawrence Reynolds returned to the Johns Hopkins Medical School, as promised, although at a later date than anticipated. He was made Instructor in Radiology with an assurance of rapid advancement on the ladder of academic promotions. As early as October, 1919, however, his quest for knowledge drove him to still newer grounds. Like the Medieval European student, who magnetized by the professors of a given institution, roved from one University to another, Dr. Reynolds was now found at the Peter Bent Brigham Hospital in Boston, where the celebrated Henry Christian and Harvey Cushing were the main attractions. From October, 1919 to May, 1922 he was Roentgenologist at that Institution and from July, 1920 to May, 1922 he was teaching as Instructor in Roentgenology at the Harvard Medical School. Yet, during these very important and fruitful years of his life, he could not forget Major Hickey, his great friend of the American Expeditionary Forces, and Preston M. Hickey could not forget the brilliant young radiologist whom he met overseas.

When, in 1922 Doctor Hickey was appointed Professor of Radiology at the University of Michigan in Ann Arbor, and abandoned the private practice of radiology, he used all his influence to persuade this brilliant young radiologist to join the organization that he founded in Detroit in 1900. It was an auspiciously fortunate day for this organization, which already had the benefit of the administrative genius of Dr. William A. Evans, that Dr.

Reynolds accepted the offer and in May, 1922 became the third member of the partnership, with the name of Drs. Hickey, Evans and Reynolds.

From 1922 until his death Dr. Reynolds practiced and was a most ardent disciple of private radiology, advantageously supported and enhanced by scientific investigations and academic teaching. His innate qualities of friendliness, graciousness, and sympathetic concern for the welfare of his patients were transcended only by his gentleness, charitableness and unbounded generosity.

After the retirement of Dr. Hickey from the partnership, the organization was known under the name of Drs. Evans and Reynolds, and, since the death of Dr. Evans in 1940, it has carried the name of Dr. Lawrence Reynolds and Associates.

Dr. Reynolds was appointed to the staff of Harper Hospital in 1922 and successively became Chief, Department of Radiology, Harper Hospital (1940); Professor of Radiology, Wayne University, College of Medicine (1945–1952); Clinical Professor of Radiology, Wayne State University, College of Medicine (1953–1959); Emeritus Professor of Radiology, Wayne State University, College of Medicine (1960–1961); and Visiting Professor of Radiology, University of Alabama Medical College (1960–1961). In addition, for many years he had been a Consultant in Radiology on the staffs of several other hospitals in Detroit.

In 1945 and from 1948 to 1959 he was Chief of The Medical Staff, Harper Hospital and Chairman, Executive Committee, Harper Hospital.

Dr. Reynolds was a member or fellow of numerous medical societies. He was Past President of the Detroit Roentgen Ray and Radium Society, Detroit Medical Club, Detroit Academy of Medicine, American Roentgen Ray Society and American College of Radiology.

Dr. Reynolds had received the honorary degrees of Doctor of Laws from the University of Alabama (1949) and the Wayne State University (1956); and the Honorary Citation of the Michigan State Medical Society (1959). He was an Honorary Member of the Radiologic Societies of Germany, Italy and Colombia. He was a Pancoast Lecturer (1941), Caldwell Lecturer (1944), Gold Medalist of the Radiological Society of North America (1956) and Gold Medalist of the American College of Radiology (1960).

The great American heritage and the uniquely cultural background of Dr. Reynolds laid the foundation, unconscious to himself in his consecration to ideals, for an inspired bibliophile, authoritative librarian and eminent scholar.

His bibliophilistic inclinations were already manifested during his assignment to the European Theater of War, when in his spare moments—as few as they were—he would prowl in sheltered book-shops along the Seine or visit fascinating galleries in more secure abodes to find some "antique books" of medical interest. These heuristic pursuits, which were assiduously followed throughout his lifetime resulted in one of the world's finest private collections

of medical texts, old original manuscripts and rare holographs. It was undoubtedly the greatest moment of his life when on February 2, 1958, in Birmingham, Alabama, at the dedication of The Lawrence Reynolds Library, which appropriately was named in his honor, he could say: "A diploma from the University of Alabama graced my wall for many years and everytime I looked at it, I felt greatly indebted; so I am returning it to you with this handful of books." This "handful of books" was a half-million dollar collection of early editions of works of some of the greatest medical and scientific pioneers of the fifteenth through the nineteenth centuries. At these impressive dedicatory ceremonies, Mr. Hill Ferguson, Chairman of the Board of Trustees, University of Alabama, presented Dr. Reynolds with a bookplate bearing the family coat of arms, which is to appear on all the books of this library.

Dr. Reynolds' inborn reverence for books made him particularly qualified to serve on library commissions with authoritative knowledge. He was an enthusiastic member, trustee or adviser of several of the famous medical libraries in various parts of the country and he was extremely proud of the fact that he was the "Library Commissioner with the longest tenure of service to the City of Detroit." On January 22, 1957, he was awarded by the late Mayor Albert E. Cobo the Citation for Meritorious Service.

In his devotion to scholarly work as writer, reviewer and editor, Dr. Reynolds exerted unbelievable steadfastness, always inspired by advancing progress through unceasing search for truth. He contributed to the medical literature numerous articles on important original investigations, countless reviews with unusually keen analytic appraisal and for over three decades he was the illustrious Editor and leading spirit of the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE. In 1956, on the occasion of the Fiftieth Anniversary of this JOURNAL he, with characteristic self-effacement, admirably described why it had become one of the leading radiologic periodicals of the world. "The founder and first Editor of the Journal, Doctor Hickey," wrote Dr. Reynolds, "blazed the trail of a high standard of scientific endeavor from which the Journal has not deviated. The Societies, the Editorial Staff, the Publisher, Mr. Charles C Thomas, and the Printer, the Banta Publishing Company, have combined their efforts in producing a scientific Journal of distinctive quality. The American Roentgen Ray Society and the American Radium Society can take justifiable pride in the splendid position which their official Journal occupies today."

Dr. Reynolds, possessed by an extremely friendly nature, approached his task graciously and accomplished it with ingenuity, vision and charm. As a famous bibliophile, authoritative librarian, eminent scholar, and foremost of all as a great radiologist, he always remained an inspiringly genial man His amazing achievements—to quote from the honorary L.L.D. citation of Wayne State University—"have helped to shape the whole course of modern radiological medicine."

TRAIAN LEUCUTIA

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Sixty-third Annual Meeting: Shoreham Hotel, Washington, D. C., October 2–5, 1962.

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Representative on the Board of Chancellors of the American College of Radiology: Milford D. Schulz, Boston, Mass.

Forty-fourth Annual Meeting: Waldorf-Astoria Hotel, New York, N. Y., April 2–4, 1962.

* *Ex officio.*

BOOKS RECEIVED

Advances in Oto-Rhino-Laryngology. Vol. IX. Edited by Prof. Dr. L. Rüedi, Zürich, Switzerland. Cloth. Price, $10.00. Pp. 323, with illustrations and tables. S. Karger, Basel, Switzerland. In the U.S.A. and Canada, Albert J. Phiebig, P. O. Box 352, White Plains, N. Y., 1961.

Carbon-14 Compounds. By John R. Catch, The Radiochemical Centre, Amersham, Buckinghamshire. Cloth. Pp. 128, with illustrations and tables. Butterworth, Inc., 7235 Wisconsin Avenue, Washington, D. C., 1961.

Formulating X-Ray Technics. Fifth edition. By John B. Cahoon, R.T., F.A.S.X.T., Assistant Professor of X-Ray Technology, Duke University Medical Center, Durham, North Carolina. Cloth. Price, $7.50. Pp. 351, with many illustrations. Duke University Press, College Station, Box 6697, Durham, N. C., 1961.

The Radiation Chemistry of Water and Aqueous Solutions. By Augustine O. Allen, Ph.D. Senior Chemist, Brookhaven National Laboratory. Cloth. Price, $6.00. Pp. 204. D. Van Nostrand Company, Inc., 120 Alexander Street, Princeton, New Jersey, 1961.

The Story of X-Rays from Röntgen to Isotopes. By Alan Ralph Bleich, B.A., M.D., Assistant Clinical Professor of Radiology, New York Medical College; Clinical Instructor of Radiology, New York University—Bellevue Medical Center, College of Medicine; and Diplomate, American Board of Radiology. Paper. Price, $1.35. Pp. 175, with 54 illustrations. Dover Publications, Inc., 180 Varick Street, New York, N. Y., 1961.

Annual Report on the Results of Treatment in Carcinoma of the Uterus. Vol. 12. Published under the patronage of the International Federation of Gynecology and Obstetrics. Cloth. Pp. 336, with many illustrations and tables. Editor: Dr. H.-L. Kottmeier, Radiumhemmet, Stockholm 60, Sweden, 1961.

Klinische Physiologie; Aktuelle Probleme in Übersichten. By Dr. W. A. Müller, Chefarzt der II. Inneren Abteilung des Robert-Bosch-Krankenhauses, Stuttgart, Germany. Cloth. Price, $4.05. Pp. 300, with tables. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U. S. A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Broncoscopia; Principi di Diagnostica e Atlante di Fotografia Endoscopica. By Luciano Cattaneo, Clinica Otorinolaringoiatrica, dell'Università di Pavia; and Lucio Di Guglielmo, Istituto di Radiologia dell'Università di Pavia. Cloth. Pp. 399, with 312 illustrations. Il Pensiero Scientifico, Editore, Roma, 1961.

Collected Papers of King Gustaf V's Jubilee Clinic, Karolinska Sjukhuset, Stockholm. Vol. 9, 1959, and Vol. 10, 1960.

The Scandinavian Journal of Clinical & Laboratory Investigation. Supplementum No. 55. An Experimental Study on Serotonin (5-Hydroxytryptamine; 5HT) on Man; with Special Reference to Renal Function. By Göran Bojs. Paper. Pp. 88 with many tables. Göteborg, 1961.

The Scandinavian Journal of Clinical & Laboratory Investigation. Supplementum No. 56. Bilirubin Determination in Serum and Urine; Studies on Diazo Methods and a New Copper-azo Pigment Method. By Magnus Michaëlsson Paper. Pp. 80, with illustrations and tables. Oslo, 1961.

The Scandinavian Journal of Clinical & Laboratory Investigation. Supplementum No. 57. Circulatory Studies in Slowly Developing Anaemias; Evans Blue Dye Dilution Curves, Roentgenologic Heart Volume Determinations, and Electrocardiographic Findings in the Anaemic State and on Recovery. By Heikki Bäckman. Paper. Pp. 127, with illustrations and tables. Mikkeli, Finland, 1961.

Unit-Step Radiography; Simplification of Medical Radiography Through Manual and Automatic Use of an Exposure Value Scale (XVS) System and Standardization. By Gerhart S. Schwarz, M.D., Department of Radiology, College of Physicians and Surgeons, Columbia University, Radiological Service, Presbyterian Hospital, New York, New York. Cloth. Price, $8.75. Pp. 230, with 33 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

An Atlas of Detailed Normal Pneumoencephalographic Anatomy. By Giovanni Di Chiro, M.D., Head of Section on Neuroradiology, National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Maryland. Cloth. Price, $19.00. Pp. 328, with 283 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Selected Papers from the Institute of Cancer Research: Royal Cancer Hospital and from the Royal Marsden Hospital, 1959. Volume 14. Published by order of the Committee of Management of the Institute and the Board of Governors of the Royal Marsden Hospital, London. Cloth. Pp. 987, with many illustrations. London, 1961.

Le Petit Cancer de L'Estomac. By J. Massa, Ancien adjoint à l'Hôpital Universitaire de Bruxelles; Diplômé en Radiologie et Electrologie de l'Université de Paris. Paper. Pp. 113, with 77 illustrations. Masson & Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris (VI^{e}), France, 1961.

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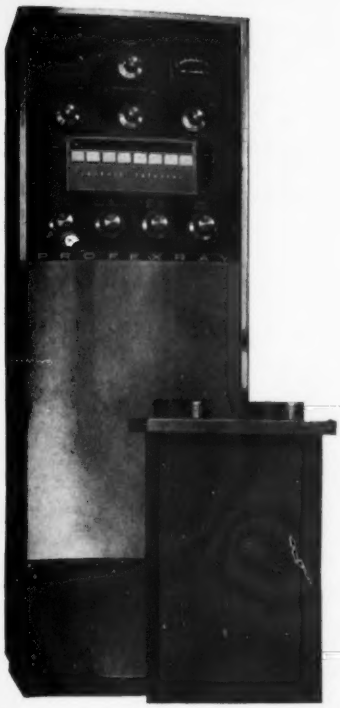

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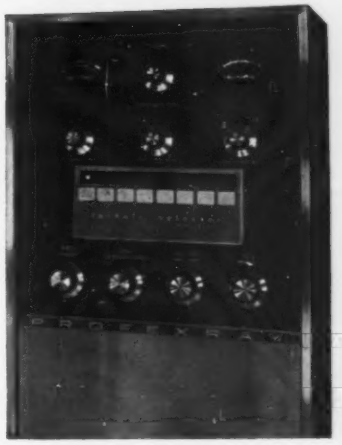

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Entered as Second-class at Springfield, Illinois under the
Act of March 3, 1879. Printed in U.S.A.

Second-class postage paid at Springfield, Illinois
and at additional mailing offices.

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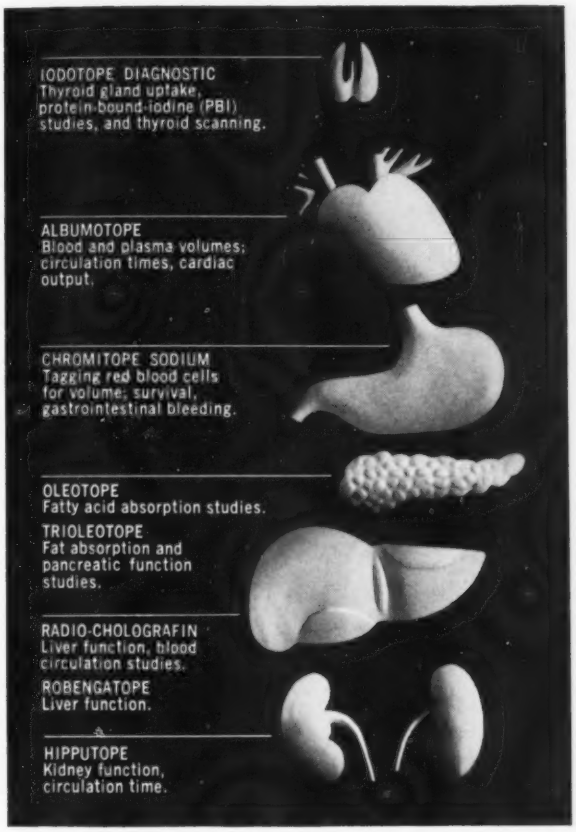

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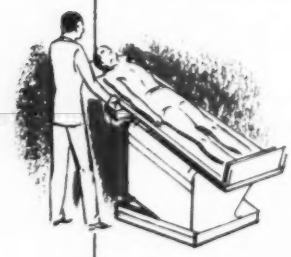

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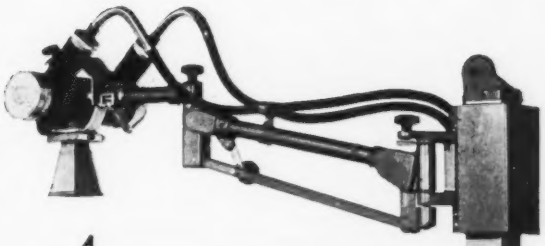

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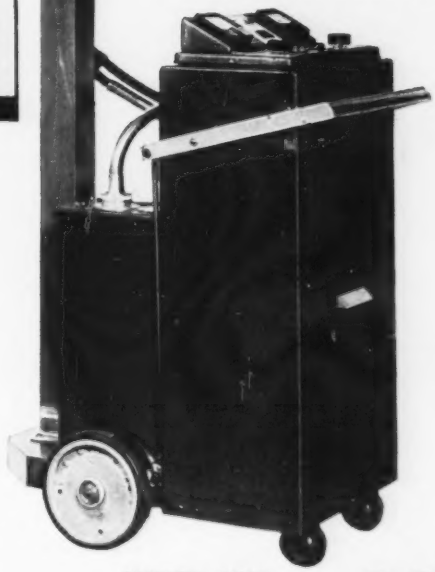

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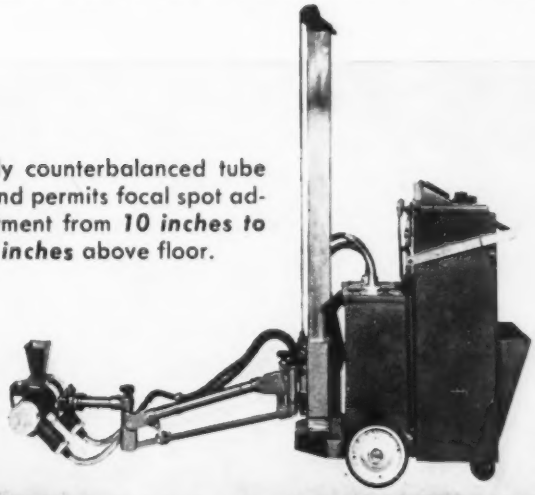

Illustration shows tube "parked" for easy moving or storage.

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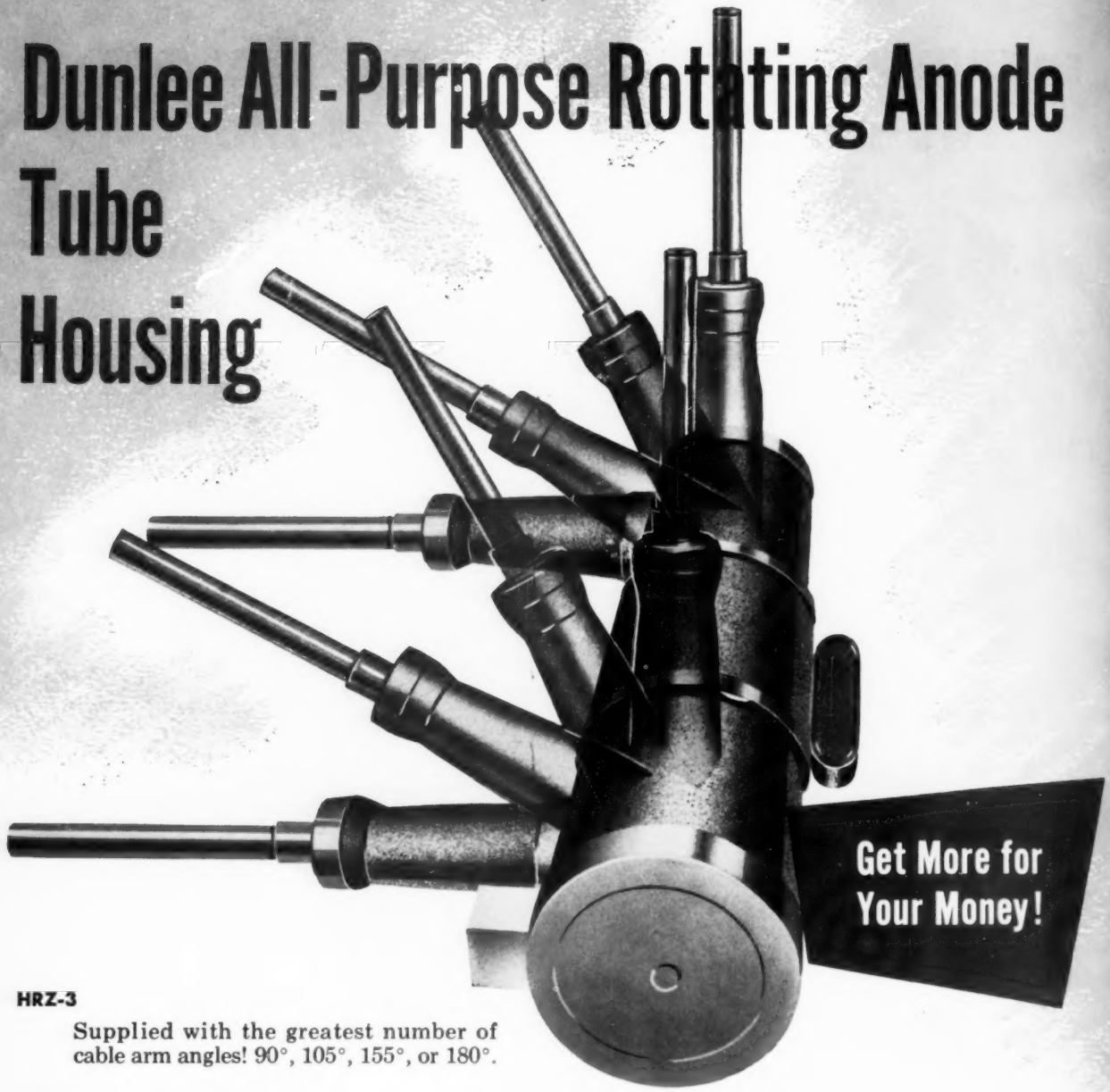

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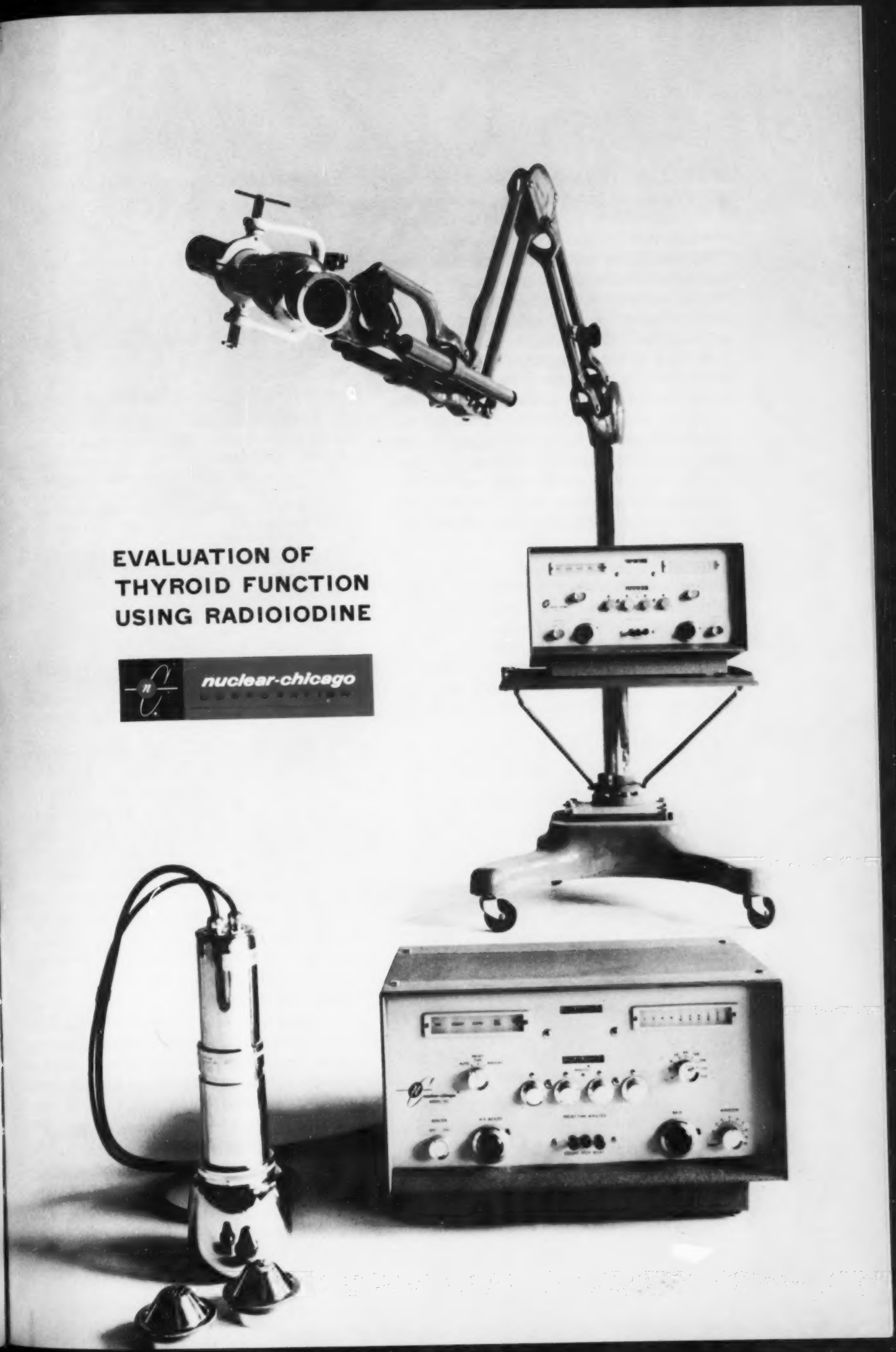

EVALUATION OF
THYROID FUNCTION
USING RADIOIODINE
nuclear-chicago

EVALUATION OF THYROID FUNCTION

The evaluation of thyroid function with radioactive iodine is the most widely used clinical application of radioisotopes. This test is based upon the well established relation between the avidity of the thyroid gland for iodine and its functional state. Iodine is used by the gland in the synthesis of the hormones thyroxin (T4) and triiodothyronine (T3) which are secreted into the blood stream for distribution to the tissues where they serve as key metabolic regulators. The rate at which the thyroid gland accumulates radioiodine from an orally administered dose and secretes labelled hormone into the blood stream are useful indexes of its function.

Of the several tests used for thyroid function, the direct measurement of iodine uptake by the thyroid gland is by far the most popular. The estimation of accumulation in the gland is usually made 24 hours after the oral administration of a tracer dose, although there is evidence that shorter time periods may be employed.

TYPICAL METHOD USING SCALER

1. Administer 5-25 microcuries of radioiodine by mouth as a solution diluted in a glass of water or as a precalibrated capsule. The material is rapidly and essentially completely absorbed from the gastrointestinal tract.
2. Set aside an identical dose of the radioiodine to be used later as a "standard." Store the standard in a small lead shield, removed from the place of counting until it is required.
3. The patient returns in 24 hours for estimation of the per cent of the administered dose of radioiodine which has been accumulated by the thyroid in the period.
4. The uptake of radioiodine in the gland is estimated by external counting with a probe scintillation detector. The procedure is to measure the radioactivity in the standard and then in the patient's thyroid. The uptake is the ratio of these two measurements.

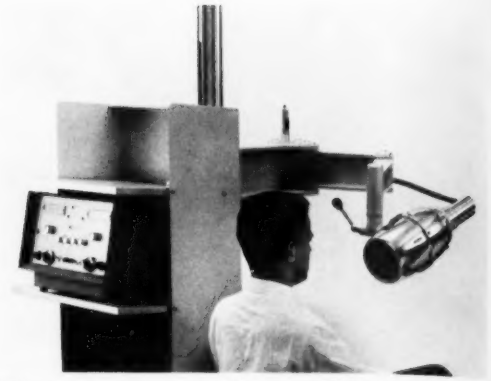


Figure 1.—Typical Nuclear-Chicago counting system for thyroid uptake measurements. Included is a Model DS-301 Scintillation Detector with 3" crystal, Model 132B Analyzer Computer, and a Clinical Stand with a single arm and instrument rack.

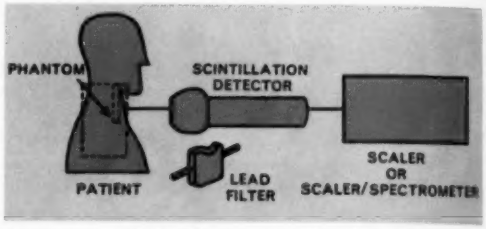


Figure 2.—Method of measuring thyroid uptake.

5. Place the standard in a plastic phantom of the neck and position it accurately along the centerline of the scintillation crystal at a distance of 25 cm. from the end of the crystal. The phantom compensates for the average distance of the thyroid under the neck surface and provides an essentially equivalent amount of absorption and scatter as the structures in the neck.
6. Determine the radioactivity in the standard at the selected distance in units of counts per minute.
7. Determine the background count by placing a 4" x 4" x ½" lead filter in front of the standard. Remove the standard and store in a lead shield at some distance from the detector.
8. Measure the radioactivity in the patient's thyroid gland at the same distance the standard was measured.
9. Measure the background count for the patient by placing the lead filter in front of the patient's thyroid gland.

10. Calculate the per cent of the administered dose of radioiodine in the thyroid gland by the formula:

$$\text{Uptake in per cent} = \frac{\text{Patient cpm} - \text{Patient background in cpm}}{\text{Standard cpm} - \text{Standard background in cpm}} \times 100$$

RESULTS

Patients with hyperthyroidism usually have uptake values between 45% and 100%. In euthyroid individuals the uptake lies between 15% and 45%. Values below 10% may be found in hypothyroidism. As in all clinical tests there are "borderline" cases which result in some overlap in the ranges given.

TYPICAL METHOD USING SPECTROMETER

The Spectrometer Method is the same as the first method except for the equipment used.

The spectrometer is used to increase the accuracy of the measurement by eliminating the scattered radiation caused by the neck and the phantom. The spectrometer is "tuned in" on the iodine-131 radiation, thus eliminating scatter and all radiations not falling within the I-131 energy band.

The spectrometer may be used two different ways: (1) It may be used to cut out all the energies both above and below the main I-131 energy by establishing a "window" around the iodine peak (See Figure 3), or (2) it may be used to eliminate only the low energy radiations below the peak (Figure 4).

ADDITIONAL TESTS AND NOTES

A confirmatory test has been developed which increases the accuracy of the radioiodine studies in cases with uptakes in the 45% range where interpretation is difficult. After the administration of 50 μg. of L-triiodothyronine (T3) for 8 days, the uptake test is repeated, taking baseline readings of radioiodine in the thyroid before giving the second tracer dose of radioiodine. In euthyroid individuals, the uptake will be depressed below 20% by the exogenous T3. In hyperthyroids, the uptake will remain above 30%.

Information may also be obtained on the status of thyroid function by estimation of the quantity of labelled hormone in the plasma after administering radioiodine. Separation of the hormone from the inorganic radioiodide can be effected by precipitating the plasma proteins with trichloroacetic acid or by absorbing the iodide by anion exchange resins. Normally less than 45 per cent of the radioiodine in the plasma of a patient receiving the isotope 24 hours previously is in the form of hormone precipitable with the plasma proteins (PBI). In hyperthyroidism, the percentage is higher because of the more rapid removal of iodide from the circulation and the faster production and turnover of hormone in the thyroid gland.

An *in vitro* estimation of thyroid function is based on the uptake of radioiodine labelled triiodothyronine (T3) by erythrocytes in a sample of the patient's blood. The saturation of plasma protein binding sites by the high levels of thyroxin in hyperthyroidism decreases their availability for T3. Thus, T3 is bound to a greater extent on erythrocytes. Over 96 per cent of normal individuals have erythrocyte uptakes below 16 per cent of the T3 incubated with a sample of blood, when the data is corrected to a hematocrit of 100 per cent. Only 5 per cent of hyperthyroids have uptakes which overlap with normals.

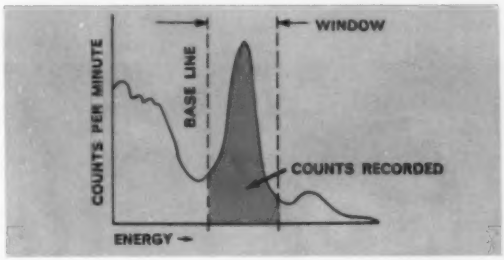


Figure 3.—Iodine-131 spectrum. Spectrometer set to count only the main I-131 energy peak.

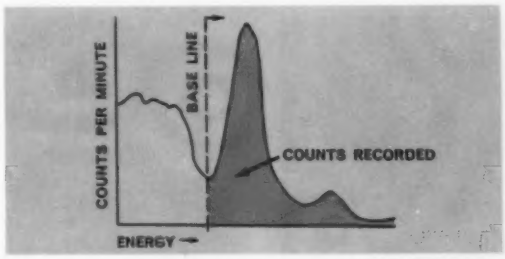
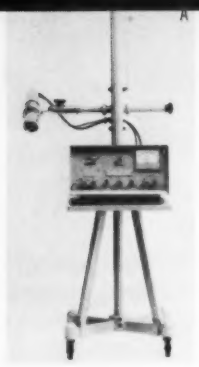
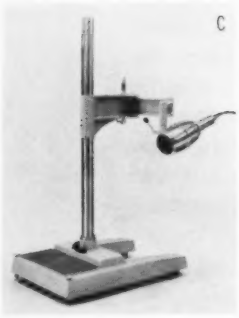
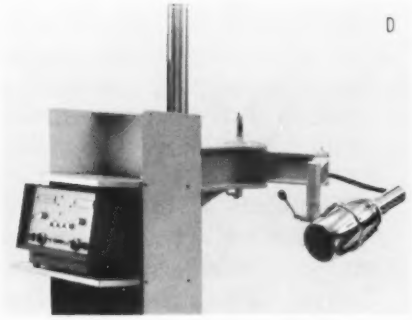
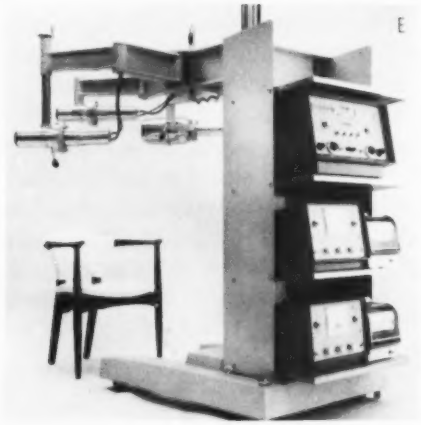


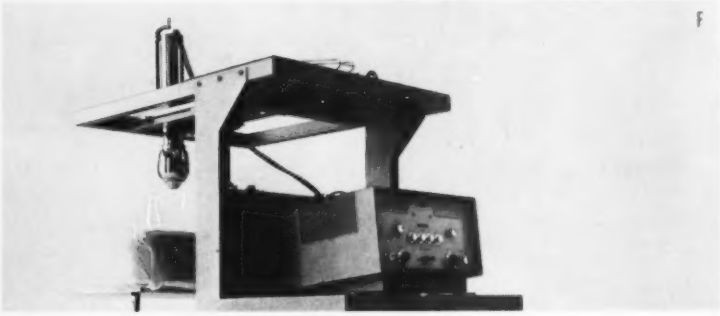
Figure 4.—Iodine-131 spectrum. Spectrometer set to eliminate low energy radiations below the iodine photo peak.

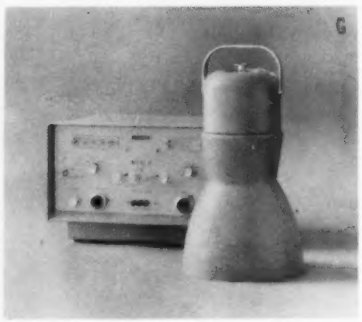












NUCLEAR-CHICAGO SYSTEMS FOR THYROID STUDIES

Pictured here are eight typical systems which Nuclear-Chicago offers the clinician for *in vivo* and *in vitro* studies of thyroid function using radioiodine. They are representative of more than 200 instruments and systems designed by Nuclear-Chicago for diagnostic medicine and research. Whatever your plans for a radiosotope program, now and in the future, there are Nuclear-Chicago instruments and systems to meet your work load and budget. You will find Nuclear-Chicago's comprehensive general catalog a valuable planning aid. Please request your copy.

A. For the modest budget, the SA1-A Detector Stand, 183B Scaler, and DS-101 Scintillation Detector with one-inch crystal are available.

B. The SA2 Detector Stand, 132B Scaler-Spectrometer, and DS-201 two-inch crystal Scintillation Detector offer higher sensitivity. The spectrometer reduces effects of scatter radiation and other sources in the vicinity.

C. Model 1710 Clinical Stand will support heavily shielded detectors such as the DS-301 with three-inch crystal. The arm is motor driven.

D. An instrument rack to hold scalers, spectrometers, ratemeters, and recorders can easily be added to the basic 1710 Stand.

E. A complete 1710 Stand includes three arms and the instrument rack. In the illustration, a three-inch detector is connected to a scaler-spectrometer for thyroid and similar studies, while two smaller detectors are connected to ratemeters and recorders for such applications as studies of kidney function and cardiac output.

F. The Isotope Scanner provides a means for scanning body areas and simultaneously producing a line scan or photographic-type record of radioisotope distribution. The system is also ideal for uptake measurements.

For *in vitro* thyroid studies, a scintillation well detector and scaler-spectrometer are used. Nuclear-Chicago offers three different well detectors. Model 132B Scaler-Spectrometer is pictured (G) with the Model DS-202 two-inch-crystal well detector and with the Model DS-302 three-inch-crystal well detector (H) which is available with a 21/32" diameter well or a 1⅛" diameter well. These detectors can also be used with any scaler or ratemeter available from Nuclear-Chicago.

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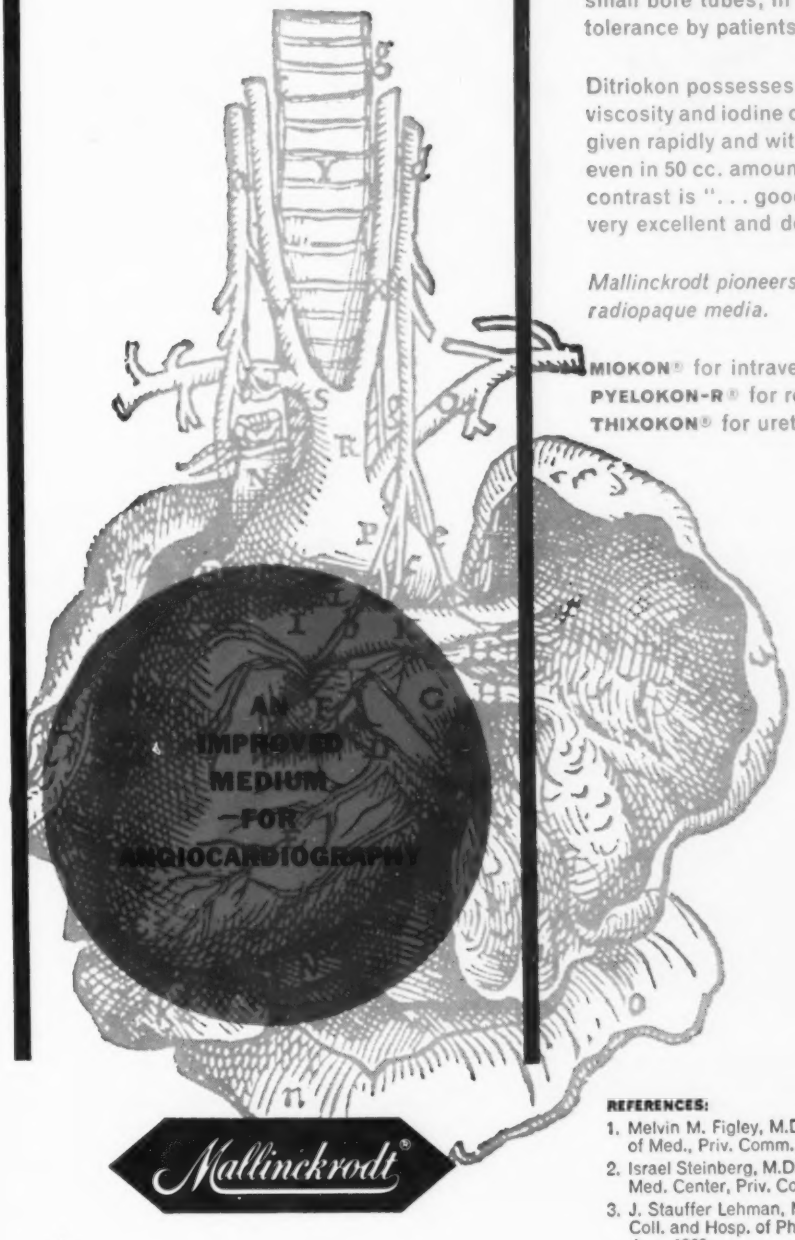

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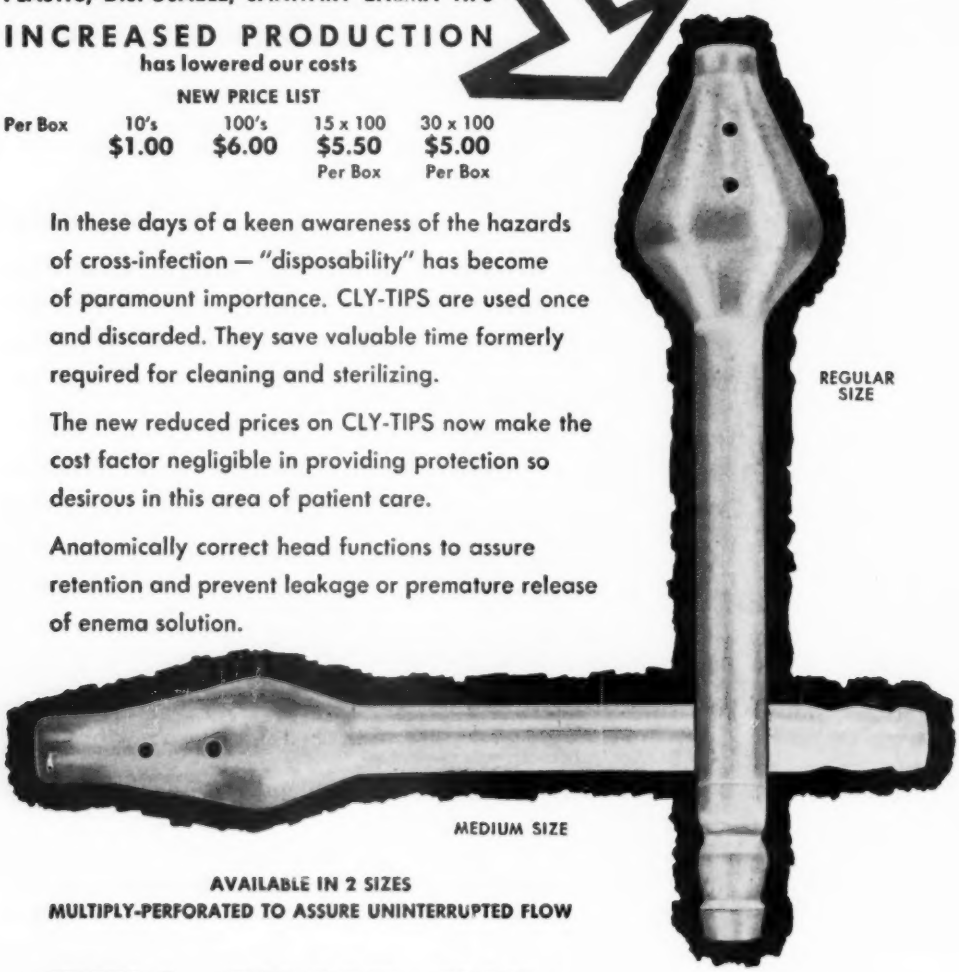

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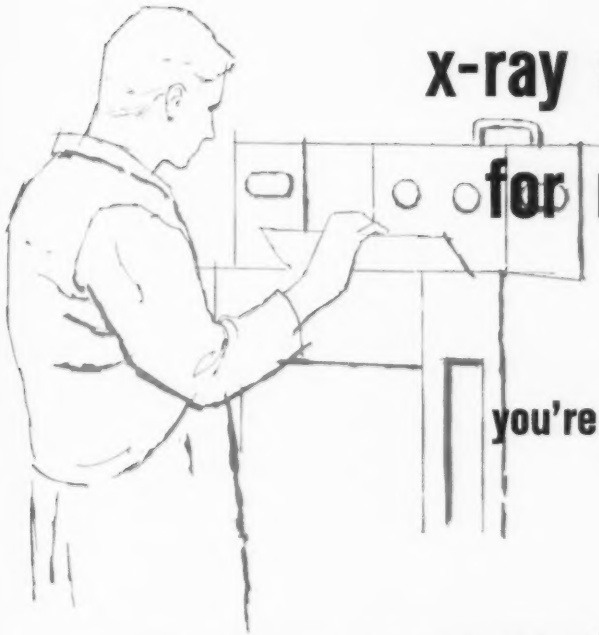

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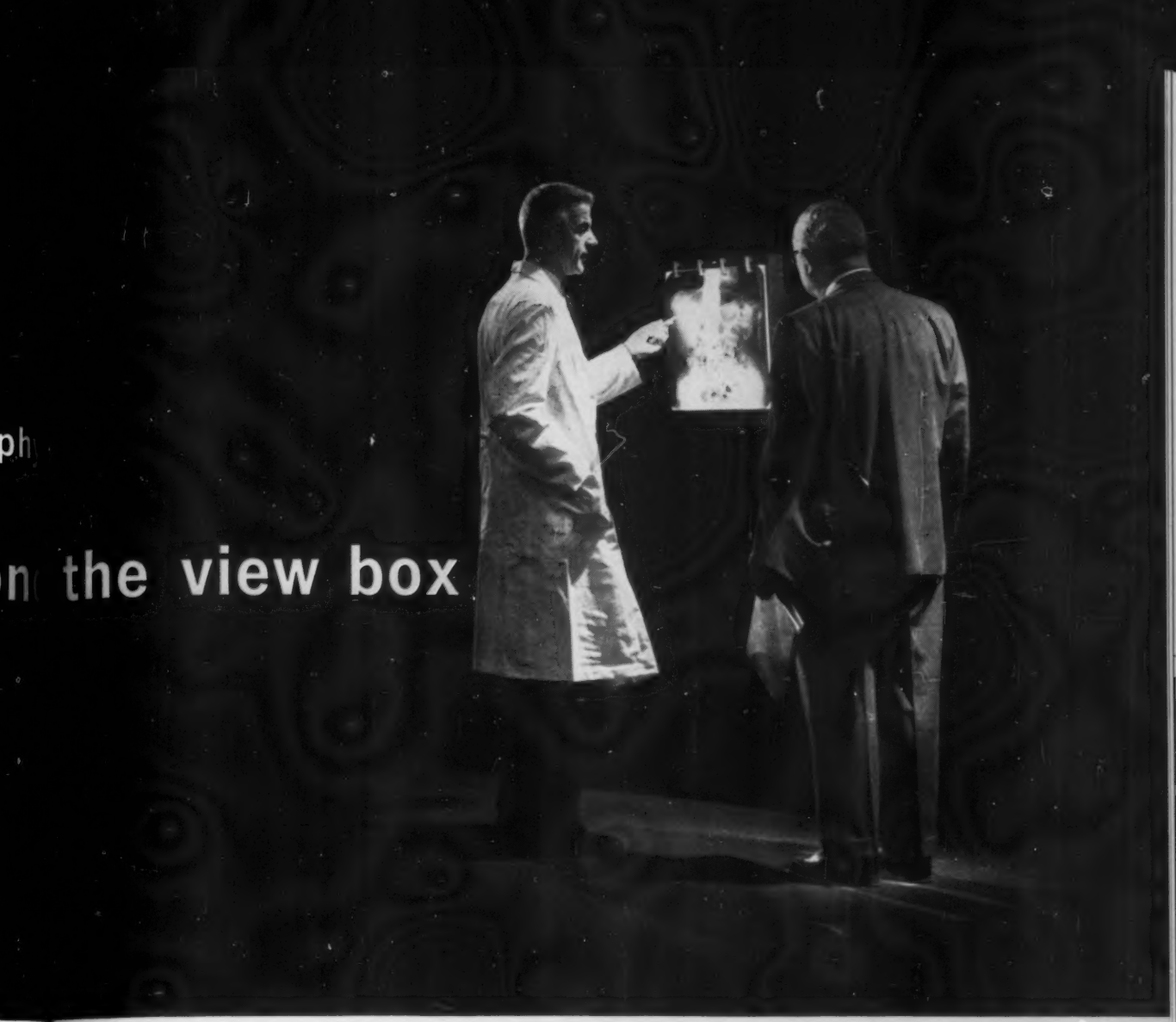

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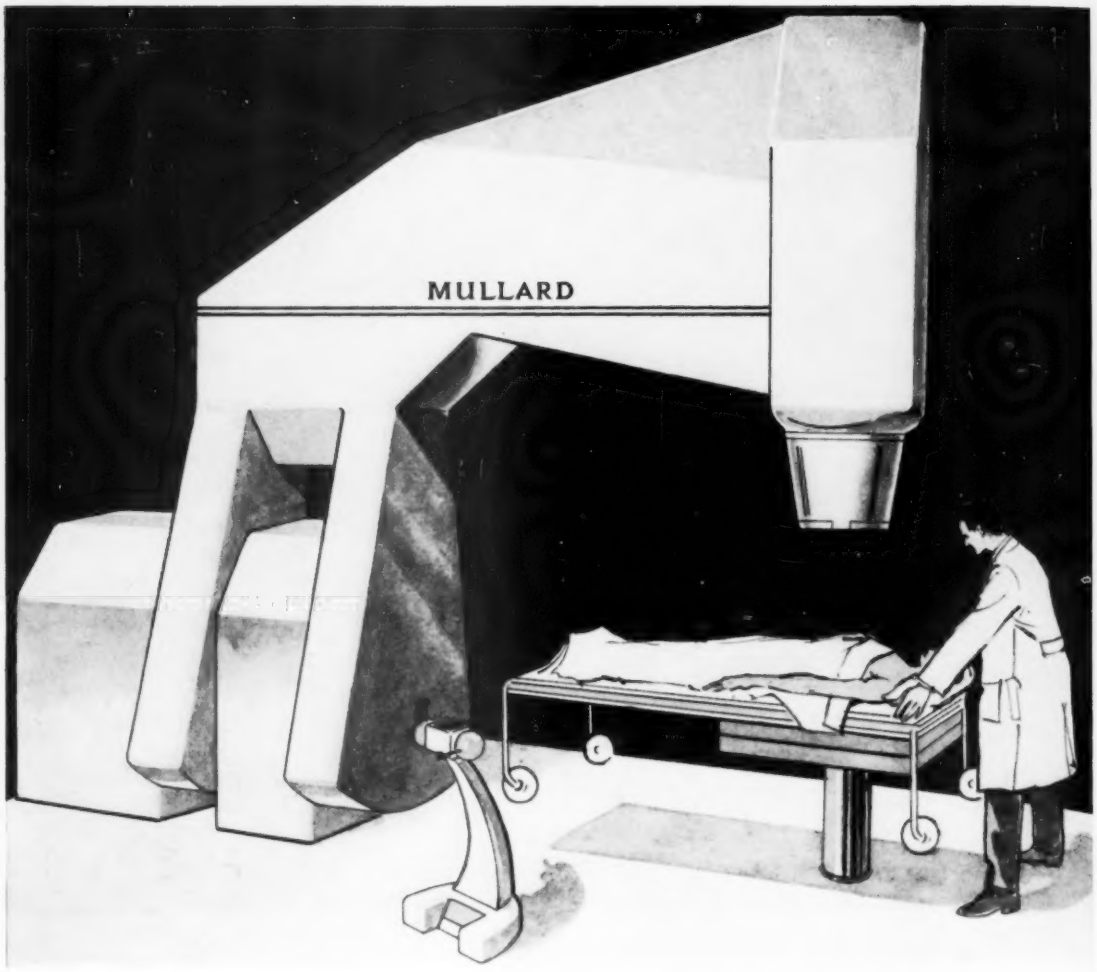

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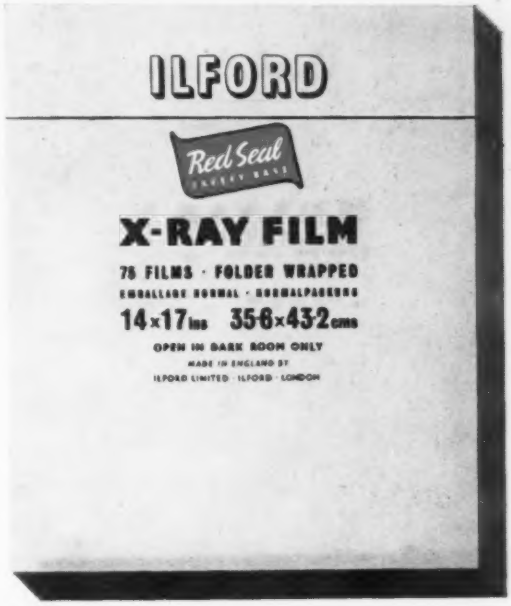

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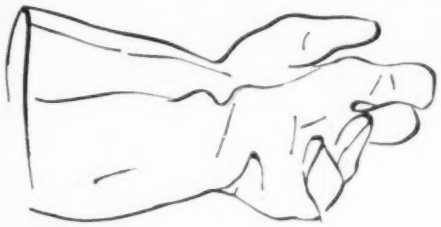

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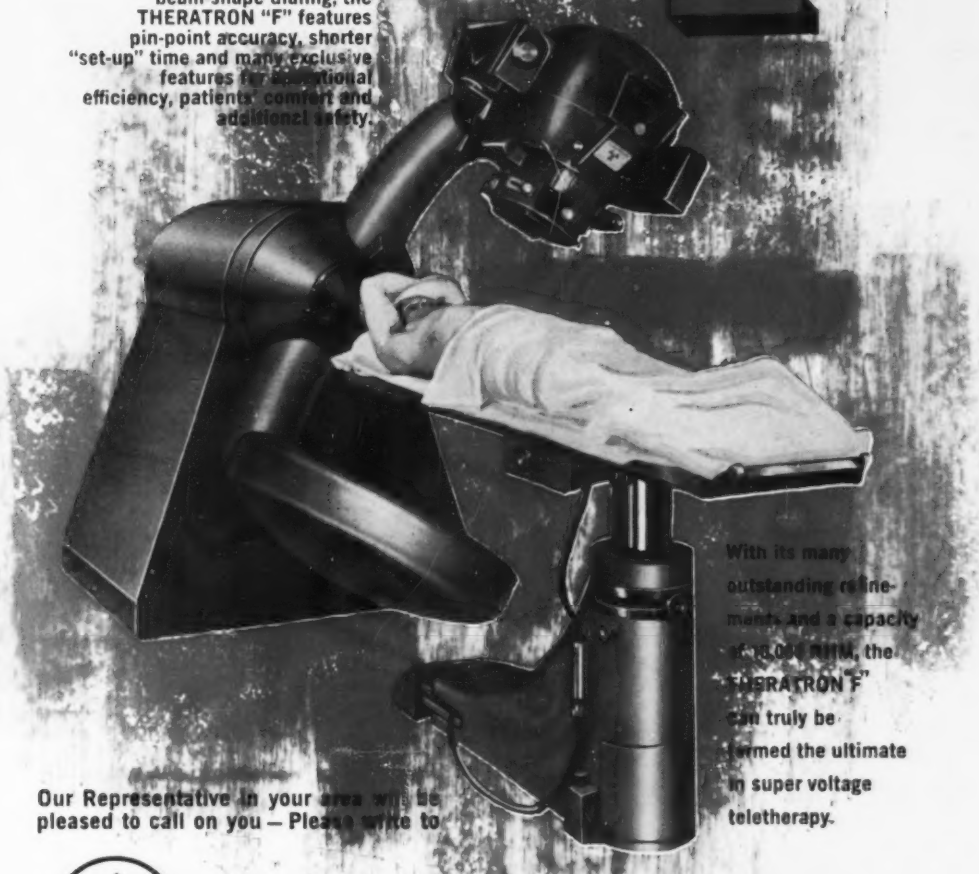

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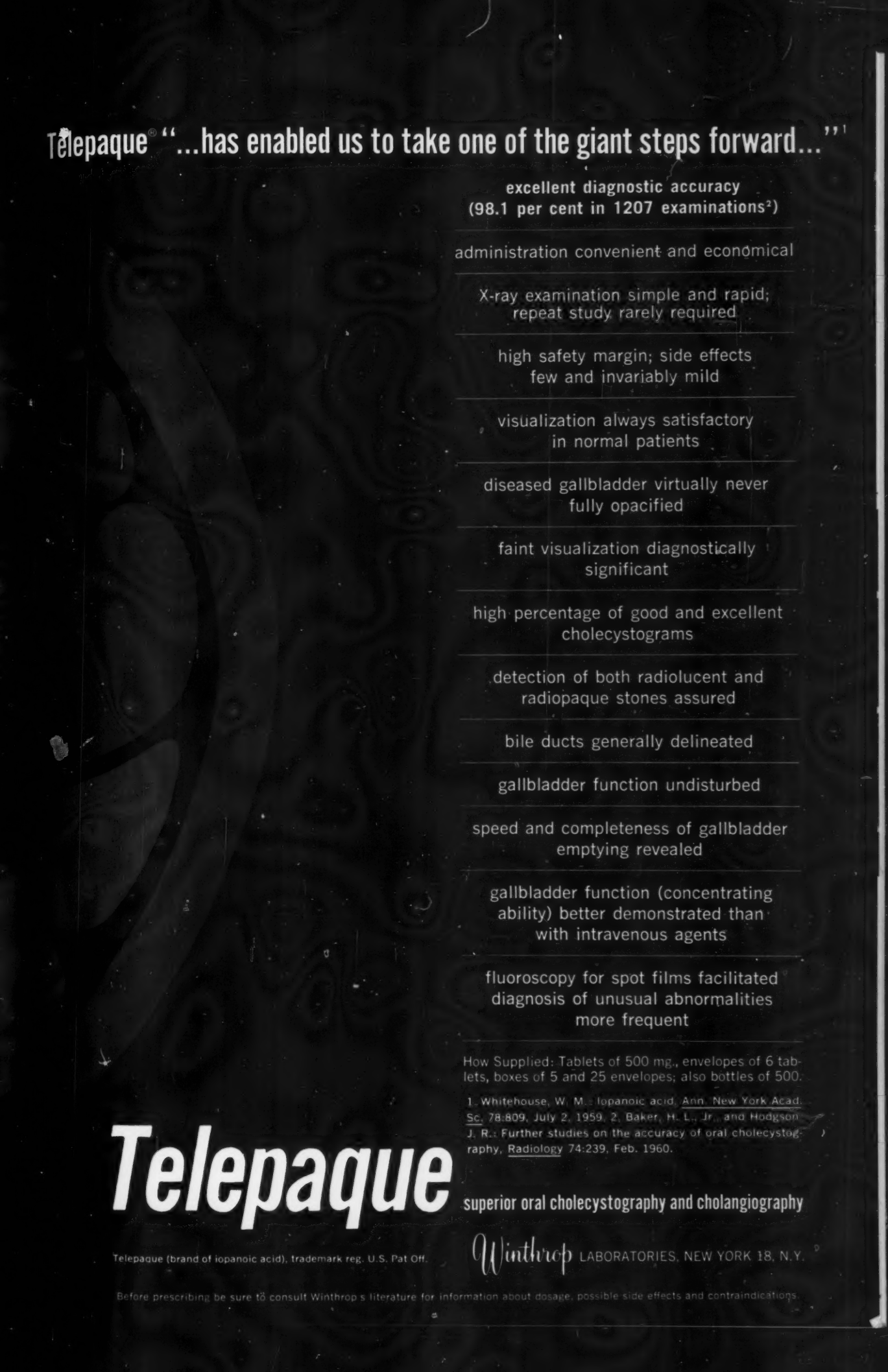

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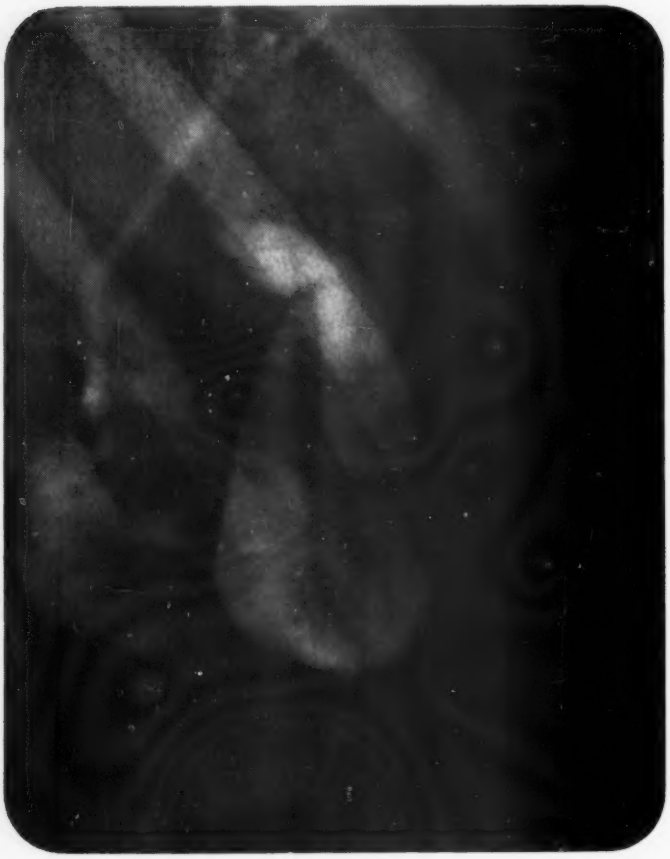

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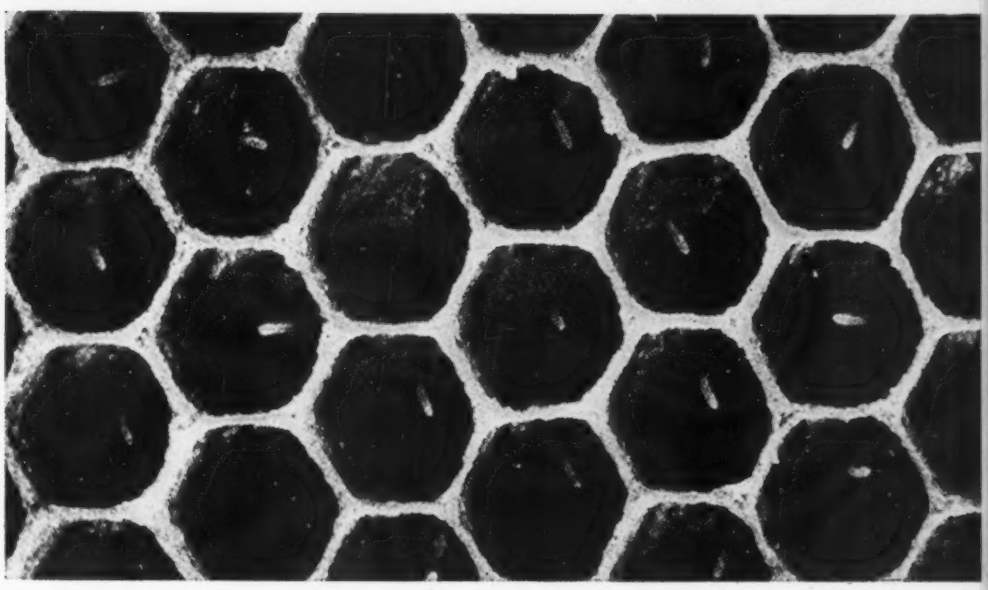

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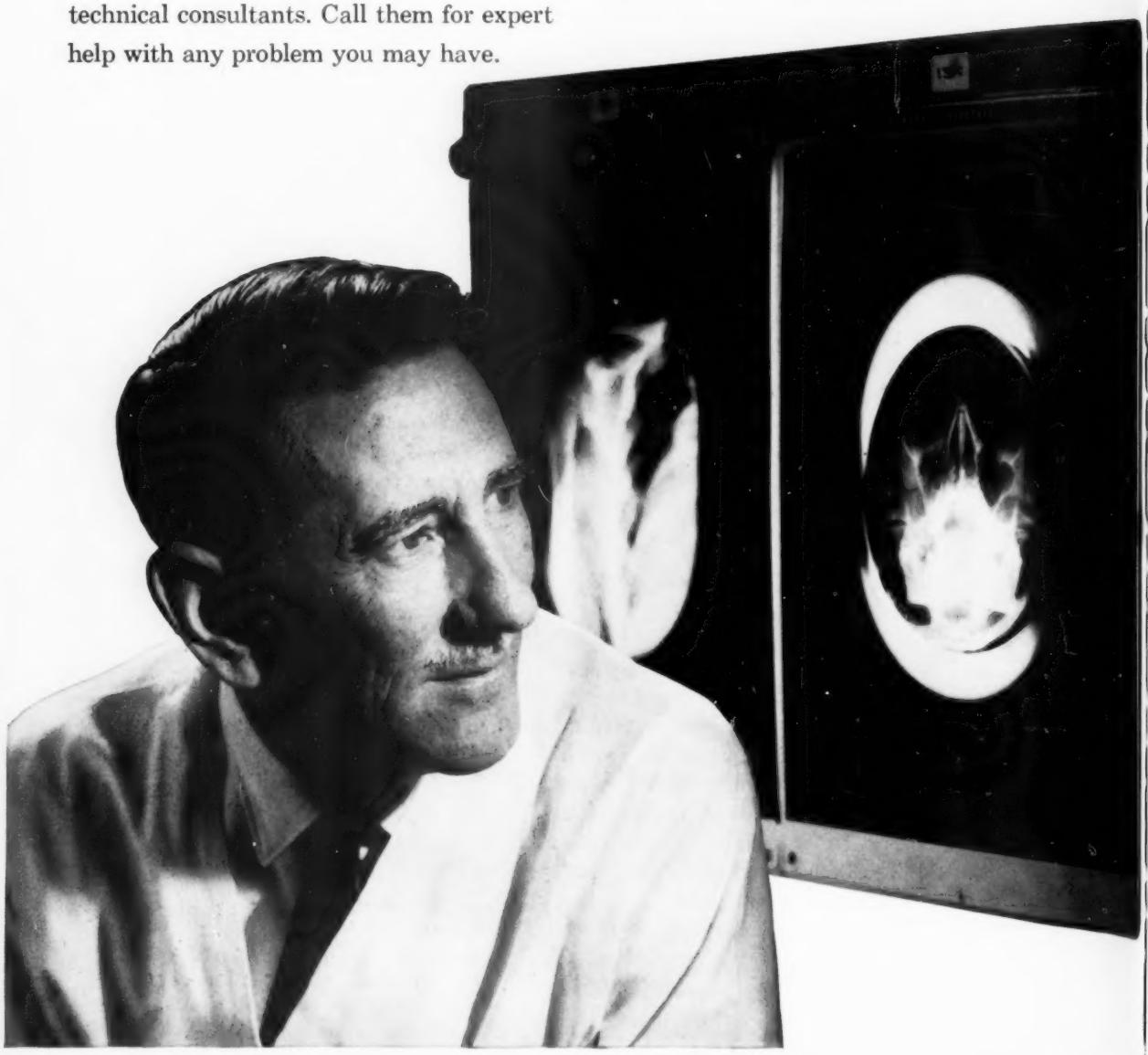

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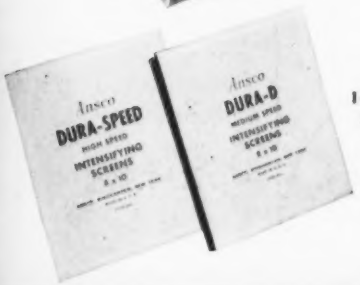
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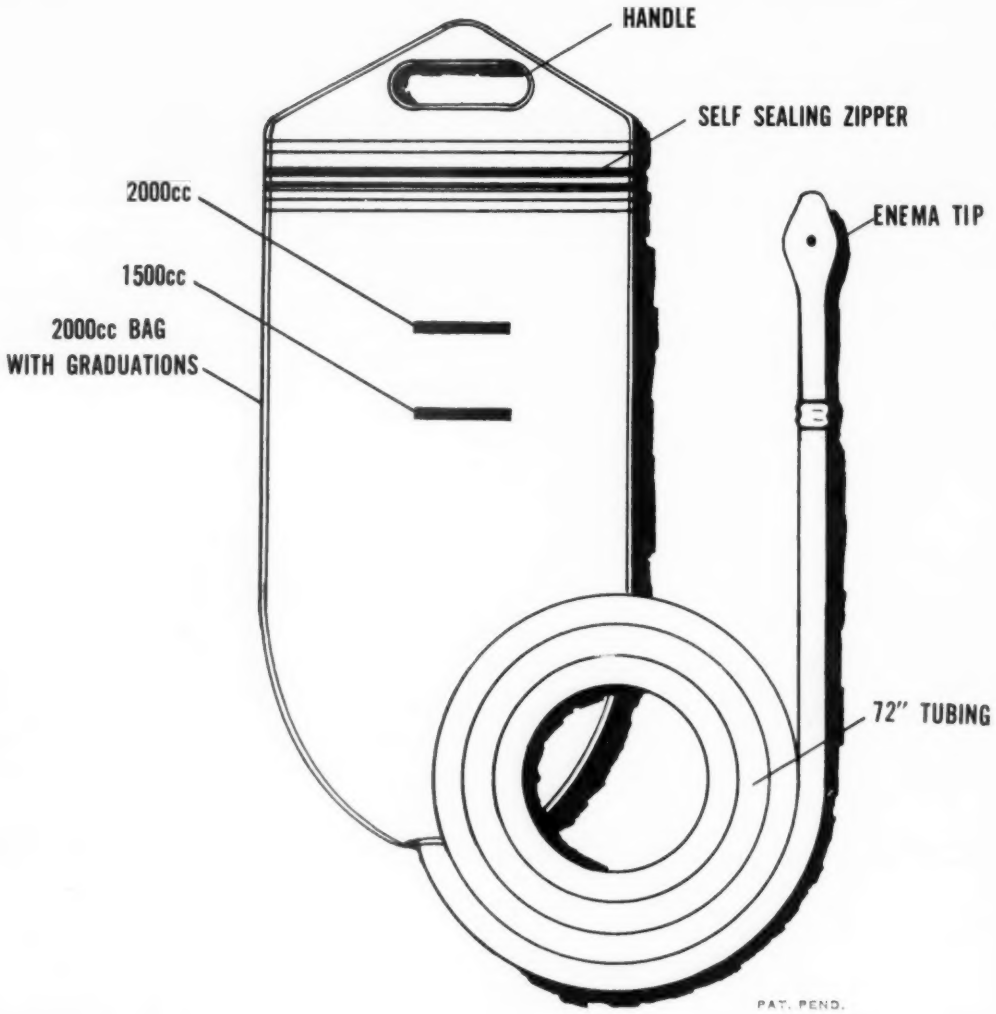

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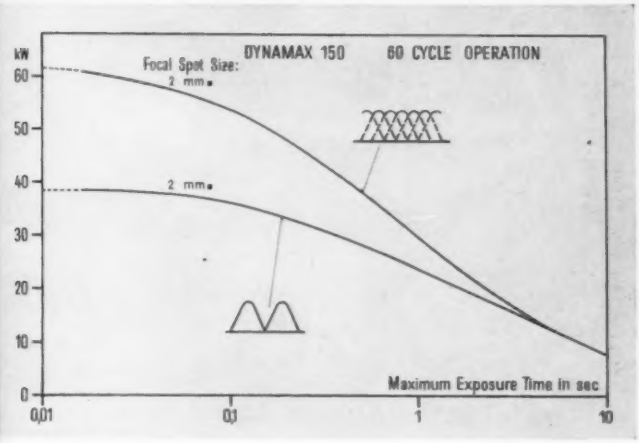

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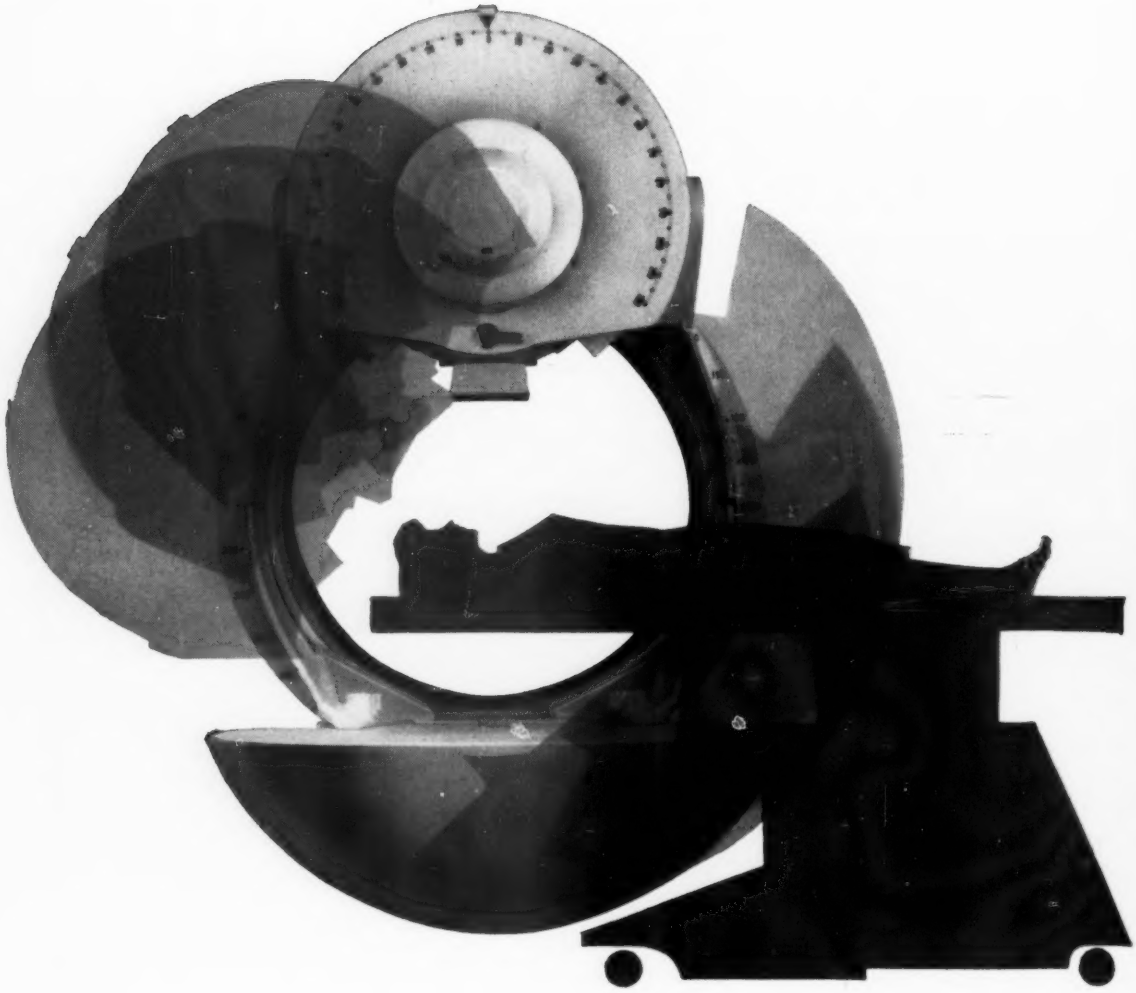

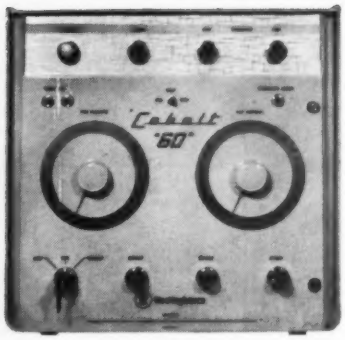

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FIGURE 1

FIGURE 2

FIGURE 3

The surgery which followed—step by step —Figure 4: Demonstrates proximal dilated loop of colon disappearing into the foramen of Winslow. *For further photographs and notes on surgery, turn page.*

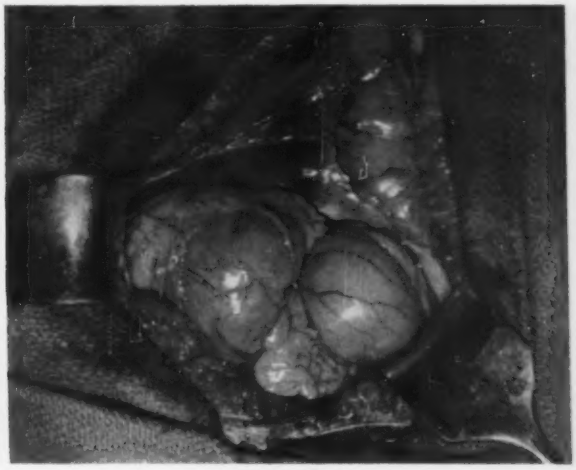

FIGURE 4

Internal hernia. *Radiographs and first step in surgery are shown on preceding page.*

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FIGURE 5

FIGURE 6

FIGURE 7

FIGURE 8

FIGURE 9

FIGURE 10

Figure 5: Appendix has been withdrawn from the lesser sac.

Figure 6: Mobile ascending colon now withdrawn from lesser sac. Note ecchymosis in mesentery which was constricted at the foramen of Winslow.

Figure 7: Demonstrates portion of omentum adherent in lesser sac.

Figure 8: Adherent portion of omentum has been sutured and divided.

Figure 9: Demonstrates extreme mobility of ascending colon with its long mesentery and constricted area in colon.

Figure 10: Foramen of Winslow has been narrowed by sutures.

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Grundlagen, Methoden und Ergebnisse der Therapie mit energiereichen Teilchen und ultraharten Strahlen

Edited by Prof. Dr. J. BECKER, Director of the Czerny-Krankenhaus für Strahlenbehandlung, University of Heidelberg, and Prof. Dr. G. SCHUBERT, Director of the Clinic of Obstetrics and Gynaecology, University of Hamburg

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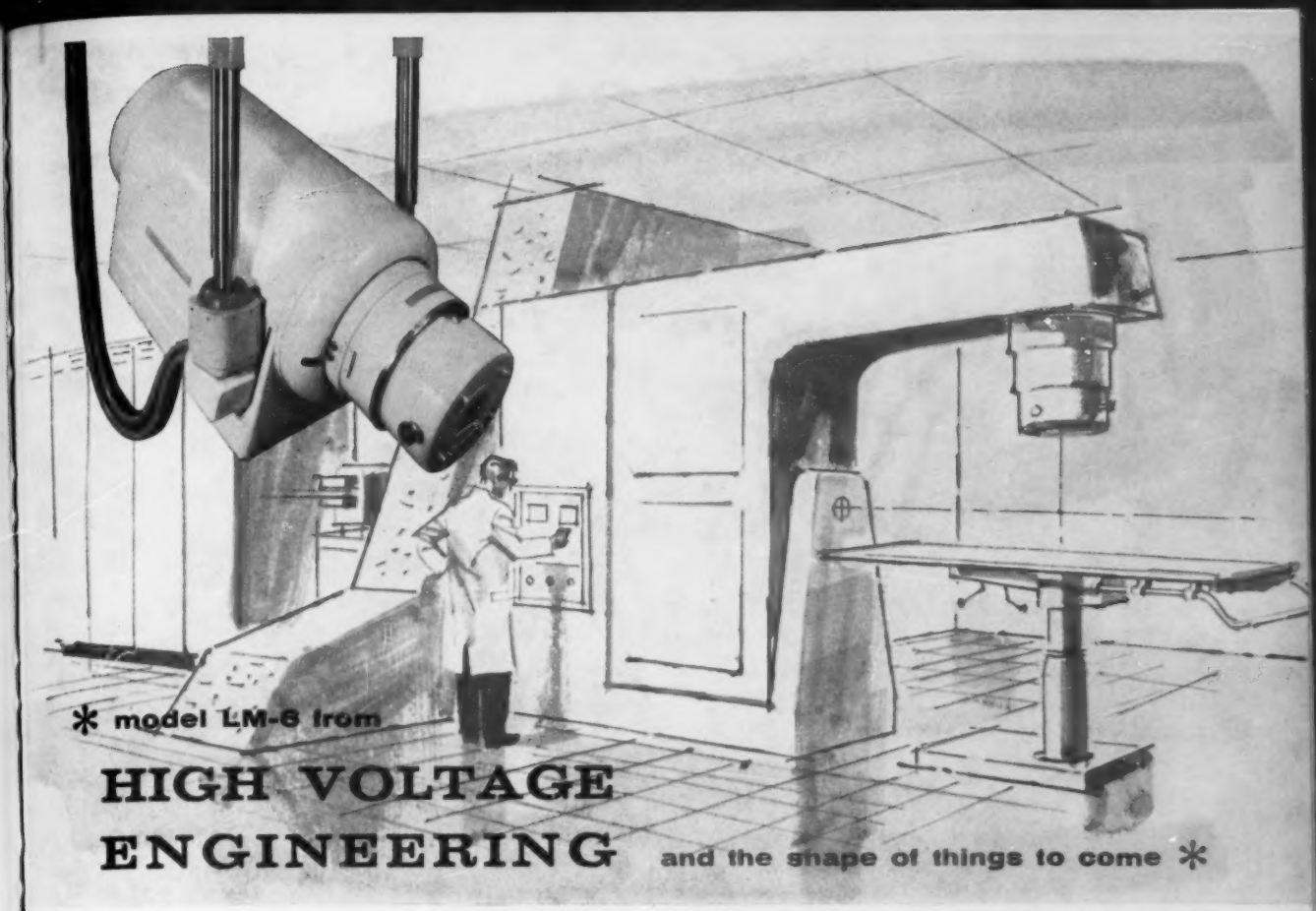

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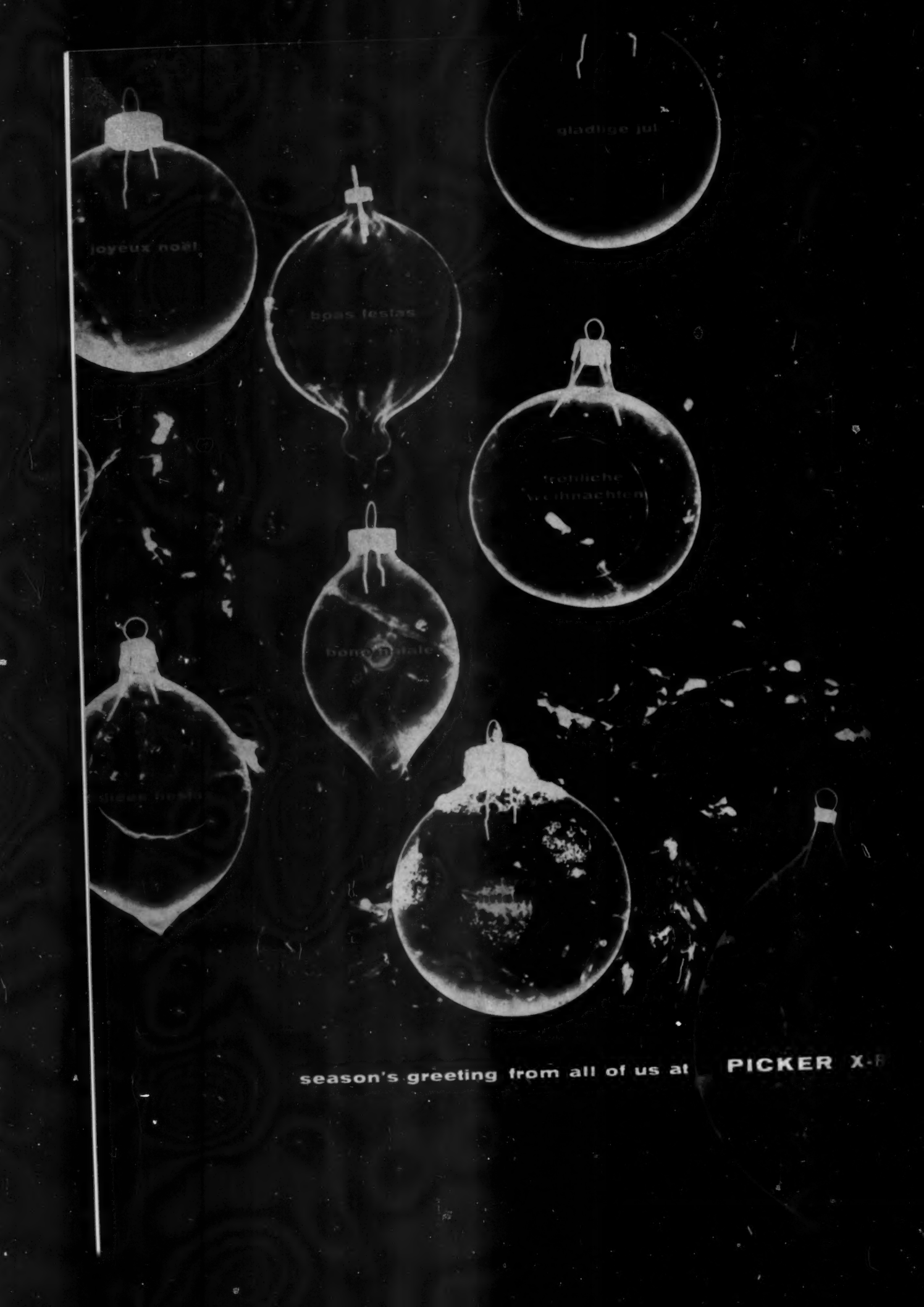
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